JOURNAL
of the
American Veterinary Medical Association

Formerly AMERICAN VETERINARY REVIEW

(Original Official Organ U. S. Vet. Med. Assn.)

H. Preston Hoskins, Secretary-Editor, 221 N. LaSalle St., Chicago, Ill.

Vol. LXXXIX, N. S. Vol. 42 — **DECEMBER, 1936** — **No. 6**

REMINDER

Letters received recently would indicate that a considerable number of state associations will take some action on the suggestion offered about a year ago, in the JOURNAL, to make membership in state associations more attractive to veterinarians in the employ of the federal government. A special committee of the National Association of Bureau of Animal Industry Veterinarians, headed by Dr. R. F. Vermilya, of Boston, Mass., has been studying the question during the past year and will report at the annual meeting of the organization in Chicago this month.

The idea of a transferable membership has been favorably received by quite a number of veterinarians in the Bureau of Animal Industry and the Army. It removes the main objection of the present requirement in most states of paying a membership fee to join each one. Veterinarians in the employ of the federal government are subject to frequent transfers. Even so, many of them would like to be affiliated with the association in the state where they are stationed, just the same as veterinarians more permanently located. Membership in the A. V. M. A. could be made a requirement and this serve in lieu of the membership fee. Or, if this is not desirable, membership in at least one state association might be required as a prerequisite.

It is understood that the secretary of each state association has been written about the proposal, with the idea that it will be discussed at the next meeting. Many of the associations will hold meetings during the winter months and we are directing attention to the matter at this time as a reminder. Recently we were asked to suggest a section that could be used as an amendment to the article covering applications for membership in the constitution and by-laws of one of the state associations. Here is what was offered:

> A veterinarian in the employ of the federal government, who is stationed in ..... and who is a member of the American Veterinary Medical Association may be admitted to active membership without the payment of the membership fee, but upon payment of the annual dues. Application for membership shall be made in the regular form prescribed in.....

See that your state association takes some action in this matter if it has not already done so.

EXECUTIVE BOARD ELECTION

The first part of the special election being held in Executive Board District 10 (Michigan and Ohio) came to a close on November 25 and the count of the ballots revealed that one Michigan member had been nominated along with four from Ohio. Every one of the five nominees is well known to the profession in District 10 and some lively balloting is expected in the election proper which is now in progress. The following is a list of the five nominees, showing their locations, connections and A. V. M. A. activities.

DISTRICT 10

GOSS, L. W. — Columbus, Ohio

Professor of Veterinary Pathology, Ohio State University. Graduate of Ohio State University, 1905. Joined A. V. M. A., 1907. Resident secretary for Kansas, 1916-19, and for Ohio, 1925-27. Member (1920-22 and 1923-24) and chairman (1922-23 and 1924-25) of Committee on Intelligence and Education; A. V. M. A. representative to American Research Council, 1921-24; chairman of Section on Education and Research, 1922-23; chairman of Committee on Policy, 1927-28; member of Committee on Poultry Diseases, 1928-29; chairman of Special Committee on Water-Borne Animal Diseases, 1929-31; member of Committee on Local Arrangements, 1935-36.

GUARD, W. F. — Columbus, Ohio

Professor of Veterinary Surgery, Ohio State University. Graduate of Ohio State University, 1912. Joined A. V. M. A., 1915. Resident secretary for Iowa, 1923-27, and for Ohio, 1930-32; 1934-. Second vice-president, 1934-35; member of Committee on Local Arrangements, 1935-36.

HILTY, REUBEN — Toledo, Ohio

Practitioner. Graduate of Ohio State University, 1907. Joined A. V. M. A., 1908. Secretary of Section on General Practice, 1917-18. Resident secretary for Ohio, 1924-25. President, 1927-28; member of Committee on Education, 1928-33; member of Special Committee on Affiliation of State and Provincial Associations, 1929-32; chairman of Committee on Resolutions, 1933-.

KILLHAM, B. J. — East Lansing, Mich.

Extension Specialist in Animal Diseases, Michigan State College. Graduate of McKillip Veterinary College, 1912. Joined A. V. M. A., 1917. Resident secretary for Michigan, 1921-22, 1924-27, 1931-33 and 1934-35; member of Committee on Local Arrangements, 1928-29; member of Committee on Veterinary Biological Products, 1929-30; member of Committee on Bang's Disease, 1930-34.

ZIMMER, F. A. — Columbus, Ohio

State Veterinarian of Ohio. Graduate of Ohio State University, 1909. Joined A. V. M. A., 1919. Member of Committee on Veterinary Biologics, 1925-26 and 1927-29; secretary of Section on Sanitary Science and Food Hygiene, 1935-36; member of Committee on Local Arrangements, 1935-36.

DOCTOR MURRAY APPOINTED DEAN

Dr. Charles Murray, who has been acting dean of the Division of Veterinary Medicine, Iowa State College, since the death of Dr. C. H. Stange in April last, was appointed dean by the Board of Education on October 30.

Dr. Murray is a graduate of Iowa State College (B. S., 1910; D. V. M., 1912) and has been connected with the institution since 1908. Before going to Ames, he attended Drake University and received the Bachelor of Pedagogy degree in 1906. In 1917, he was appointed head of the Department of Veterinary Research and has been in direct charge of veterinary research work at Ames ever since.

VERMONT TUBERCULOSIS-FREE

Vermont was added to the list of tuberculosis-free states on November 2, 1936, bringing the number up to 42. All of the New England States are now in the modified accredited area.

The Green Mountain State was a pioneer in tuberculosis eradication, work along this line having been started in the early 90's, shortly after the discovery of tuberculin. This early work was done by the late Dr. F. A. Rich, of the Vermont Agricultural Experiment Station, who published a bulletin on the subject in 1894.

Coöperative work by the federal and state governments was commenced in 1917. At first, the testing was conducted on the individual-herd basis. Later, the area plan was adopted. In July, 1932, Lamoille and Orange counties were accredited.

Only six states now remain to be cleaned up: California, South Dakota, New York, Pennsylvania, New Jersey and Maryland. Pennsylvania may be on the accredited list by the time this appears in print.

CHANGE IN REGULATIONS COVERING DOG FOODS

Some six months ago, editorial comment was made on certain developments in the canned dog food industry. Among other things the statement was made that it would appear to have been a tactical error for the manufacturers of certain dog foods to advertise that their products were "fit for human consumption." The past month, the Bureau of Animal Industry of the U. S. Department of Agriculture announced that it had been necessary to modify the regulations pertaining to dog foods and similar products.

Designated as Amendment 10 to B. A. I. Order 211, the new requirements provide for the withdrawal of the familiar meat inspection stamp from containers of dog food, cat food and other products of a similar nature. The new regulation provides, however, that containers of such products may bear the statement: "The meat or meat by-product ingredient of this article has been examined and passed under federal supervision. This article has been prepared in an establishment operating under federal meat inspection."

Amendment 10, which is now in effect, also provides that the regular meat inspection label as used on food intended for human consumption may *not* be used on food designated as being intended for dogs, cats, foxes and similar animals. Thus the inspection legend, "U. S. Inspected and Passed by the Department of Agriculture," is reserved exclusively for foods intended for *human* consumption. The regulation further provides that when dog food, or any similar product, is prepared in a part of a federally inspected establishment, it will receive the same supervision as other parts of the establishment.

The federal authorities have directed attention to the fact that the purpose of the new regulation is to inform the public that the meat ingredient has been inspected and passed, but that inspection has not included various other ingredients with which the meat has been combined.

Arrangements have been made for the Department of Agriculture to receive reports relating to the commercial output of these products, to be used for statistical purposes.

APPLICATIONS FOR MEMBERSHIP

Ordinarily November is the lightest month in the year, as far as applications for membership are concerned. However, the month just closed broke all existing records by a large margin. In November, 1928, there were 17 applications filed, establishing a record. The past month we received 42 applications, which are given first listing this month. By way of contrast, the average number received during November over a period of twelve years (1924 to 1935 inclusive) has been about seven. The total for the past three months is 115, which is more than were filed during the entire year of 1934. We have now had two record-breaking months in succession. Let's make December another one.

(See July, 1936, JOURNAL)

FIRST LISTING

APPLEGATE, RALPH W. 200 Morgan St., Tampa, Fla.
D. V. M., Cincinnati Veterinary College, 1912
Vouchers: Col. Robert J. Foster and John R. Wells.

ARMFIELD, FRANK P. Marianna, Fla.
D. V. M., Ohio State University, 1929
Vouchers: Col. Robert J. Foster and T. W. Cole.

BILD, CHARLES E. 2635 N. W. 36th St., Miami, Fla.
D. V. M., Iowa State College, 1933
Vouchers: J. H. Yarborough and John R. Wells.

BROWN, LT. ROBERT J. Akron, Ohio
D. V. M., Iowa State College, 1936
Vouchers: H. D. Bergman and Chas. Murray.

CLARK, ANDREW F. Box 617, Sarasota, Fla.
D. V. M., Alabama Polytechnic Institute, 1936
Vouchers: B. N. Lauderdale and T. W. Cole.

CLARVOE, HAROLD M. 405 S. Howard Ave., Tampa, Fla.
D. V. M., United States College of Veterinary Surgeons, 1918
Vouchers: Col. Robert J. Foster and John R. Wells.

DILTS, CHARLES R. 5707 Nebraska Ave., Tampa, Fla.
D. V. M., Ohio State University, 1904
Vouchers: Col. Robert J. Foster and T. W. Cole.

GILLIS, DEWITT C. State Live Stock Sanitary Board, Tallahassee, Fla.
D. V. M., Iowa State College, 1912
Vouchers: Col. Robert J. Foster and John R. Wells.

HAMMAN, CAPT. FRED. I. Hdqrs. Casper Dist., C. C. C., Casper, Wyo.
D. V. M., Kansas City Veterinary College, 1917
Vouchers: Albert B. Kight and Lt. Col. Geo. W. Brower.

HARTZELL, LT. HAROLD P. Fort Douglas, Utah
D. V. M., Kansas State College, 1932
Vouchers: Ernest L. Henkel and Maj. John W. Miner.

HEISHMAN, J. O. Station F, Jacksonville, Fla.
B. V. Sc., Ontario Veterinary College, 1934
Vouchers: T. W. Cole and T. H. Applewhite.

HERMAN, SAMUEL E. 120 E. 59th St., New York, N. Y.
D. V. M., Cornell University, 1935
Vouchers: Frank Bloom and M. P. Lawrence.

HOON, LT. HENRY R. 807 10th Ave., Lewiston, Idaho
B. S., D. V. M., State College of Washington, 1928
Vouchers: H. A. Trippeer and C. A. Johnston.

HUGHES, HARBARD C. 532 W. Miami Rd., Jacksonville, Fla.
D. V. M., Kansas City Veterinary College, 1912
Vouchers: T. W. Cole and John R. Wells.

HYSLOP, HERMAN T. Burns & Col., Ltd., Prince Albert, Sask.
B. V. Sc., Ontario Veterinary College, 1913
Vouchers: R. T. Skelton and R. C. Duthie.

JOHNSON, RAYMOND W. 1618 S. 24th St., Lincoln, Neb.
D. V. M., Iowa State College, 1932
Vouchers: E. C. Jones and Carl J. Norden.

JOHNSON, SAMUEL T. 816 Broad St., Jacksonville, Fla.
D. V. M., University of Georgia, 1933
Vouchers: T. W. Cole and John R. Wells.

KENNEDY, EARL R. 3715 5th Ave., Moline, Ill.
D. V. M., Terre Haute Veterinary College, 1915
Vouchers: D. M. Campbell and L. A. Merillat.

LANCASTER, LT. HARRY R. Box 776, Tuscaloosa, Ala.
D. V. M., Colorado State College, 1935
Vouchers: Col. B. A. Seeley and Roy Avant.

MCGINNIS, LT. VELMER W. Army Veterinary School, Army Medical Center, Washington, D. C.
D. V. M., Kansas State College, 1933
Vouchers: Lt. Col. Jean R. Underwood and R. R. Dykstra.

MARTIN, WALTER D. JR. 2635 N. W. 36th St., Miami, Fla.
D. V. M., Alabama Polytechnic Institute, 1934
Vouchers: J. H. Yarborough and John R. Wells.

MILLER, HERBERT E. Box 404, Coral Gables, Fla.
D. V. M., Ohio State University, 1916
Vouchers: Col. Robert J. Foster and J. H. Yarborough.

MORGAN, LT. DONALD R. 402 E. 29th St., Vancouver, Wash.
B. S., D. V. M., State College of Washington, 1930
Vouchers: Hilton A. Smith and N. G. Covington.

OMDALEN, R. O. 6150 Greenwood Ave., Chicago, Ill.
D. V. M., Ohio State University, 1933
Vouchers: G. S. Elwood and O. Norling-Christensen.

REINECCIUS, LT. JAKE L. Fort Snelling, Minn.
D. V. M., Kansas State College, 1933
Vouchers: Lt. Col. Jesse D. Derrick and Maj. J. G. Fuller.

RIPPETOE, LT. CULVER W. Hdqrs. Missouri-Kansas Dist., C. C. C., Fort Leavenworth, Kan.
D. V. M., Kansas State College, 1934
Vouchers: Lt. Col. Jesse D. Derrick and H. L. Morrison.

SELKIN, WILLIAM J. 604 E. Gun Hill Rd., Bronx, New York, N. Y.
D. V. M., Cornell University, 1913
Vouchers: Charles Hoefle and Charles V. Noback.

SMIT, LT. CHARLES R. Fort Snelling, Minn.
D. V. M., Iowa State College, 1934
Vouchers: F. W. Crawford and Maj. J. G. Fuller.

SMIT, LT. WALTER 230 5th Ave., Leavenworth, Kan.
D. V. M., Iowa State College, 1930
Vouchers: Lt. Col. Jesse D. Derrick and Maj. J. G. Fuller.

SPENCER, ARTHUR H. Lake Worth, Fla.
V. M. D., University of Pennsylvania, 1904
Vouchers: Col. Robert J. Foster and John R. Wells.

TANNER, WALLACE J. 2117 4th St., Saint Petersburg, Fla.
D. V. M., Cincinnati Veterinary College, 1912
Vouchers: Col. Robert J. Foster and John R. Wells.

THOMPSON, LT. WILLIAM M. 1203 White Ave., Grand Junction, Colo.
D. V. M., Texas A. & M. College, 1935
Vouchers: A. P. Drew and Lt. Col. Geo. H. Koon.

TODD, LT. F. ARNOLD Camp Charles M. Smith No. 11064, Waterbury, Vt.
D. V. M., Iowa State College, 1933
C. P. H., Yale University, 1935
Vouchers: Maj. Burlin C. Bridges and L. H. Adams.

UTLEY, THOMAS E. 1640 N. W. 14th St., Oklahoma City, Okla.
D. V. M., Colorado State College, 1936
Vouchers: L. J. Allen and C. H. Fauks.

WANN, LT. RUSSELL S. 2203 Jackson St., Alexandria, La.
D. V. M., Alabama Polytechnic Institute, 1934
Vouchers: Col. B. A. Seeley and I. S. McAdory.

WHITE, LT. ALFRED E. JR. 633 Chester St., Glendale, Calif.
D. V. M., Kansas State College, 1935
Vouchers: Ernest R. Sparks and Wm. R. Kermen.

WILLIAMSON, ARTHUR H. State Board of Health, Jacksonville, Fla.
B. S., Alabama Polytechnic Institute, 1921
D. V. M., Alabama Polytechnic Institute, 1924
Vouchers: Col. Robert J. Foster and John R. Wells.

WILSON, CAPT. WILLIS Dayton, Wash.
B. S., D. V. M., State College of Washington, 1909
Vouchers: J. A. Zimmerman and Col. A. L. Mason.

WINTER, EDWARD F. 210 S. Hanna St., Tampa, Fla.
D. V. M., Indiana Veterinary College, 1932
Vouchers: Col. Robert J. Foster and John R. Wells.

WIRTHLIN, JOHN R. 6002 Suwanee Ave., Tampa, Fla.
D. V. M., Cincinnati Veterinary College, 1917
Vouchers: T. W. Cole and T. H. Applewhite.

WISWELL, LT. WILBUR H.
Veterinary Station Hospital, Fort Des Moines, Iowa
D. V. M., Kansas State College, 1935
Vouchers: Lt. Col. Jesse D. Derrick and C. C. Hall.

ZEDLITZ, LT. ALFRED C. 1203 8th St., Ballinger, Texas
D. V. M., Texas A. & M. College, 1936
Vouchers: Capt. M. Shipley and W. R. McCuistion.

Applications Pending

SECOND LISTING

(See November, 1936, JOURNAL)

Adan, Lt. Cirilo, Fort Meade, S. Dak.
Allen, Lt. John K., 2555 Bryant Ave. S., Minneapolis, Minn.
Andres, Leo E., Remington, Ind.
Andrews, Capt. Asa R., Presidio of San Francisco, Calif.
Asbill, Lt. Stephen G., 302 A. St., Davis, Calif.
Bertz, Capt. Wesley W., Carlisle Barracks, Pa.
Broad, Capt. Fay E., 802 N. Michigan, Plymouth, Ind.
Cairy, Lt. Clyde F., 4900 Morningside Ave., Sioux City, Iowa.
Carll, Lt. Walter T., Fort Hoyle, Md.
Castleberry, Capt. Guy, 1140 E. 8th South St., Salt Lake City, Utah.
Chastain, Capt. Walter R., Station F, Box 3192, Jacksonville, Fla.
Cruse, Charles L., 906 Northwest Blvd., Winston-Salem, N. C.
Deal, Lt. Alfred F., C. C. C. Camp S-71, Lake Placid, N. Y.

Dobson, Charles C., New Augusta, Ind.
Ellis, Capt. Harvie R., Fort Riley, Kan.
Estes, Robert F., Orange, Va.
Gerry, Russell W., 8474 Melrose Ave., West Hollywood, Calif.
Greene, Lt. James E., 107 E. John Calvin St., College Park, Ga.
Harter, William L., 351 N. Foothill Rd., Beverly Hills, Calif.
Harvey, Marvin H., 9088 Santa Monica Blvd., West Hollywood, Calif.
Hibbs, Lt. Leonard W., 927 Cleveland, Kansas City, Kan.
Higby, Lt. Willard C., Turin, N. Y.
Hock, Capt. Leo A., 725 Thurman Ave., Columbus, Ohio.
Huber, Lt. Samuel F. Jr., Y. M. C. A., Schenectady, N. Y.
Ishee, Lt. Vaughn E., Veterinary Station Hospital, Fort Knox, Ky.
Kelley, Lt. Donald C., 20-21 Arnold Hall, Fort Riley, Kan.
Kerr, Virgil M., Box 1086, Sacramento, Calif.
Kielsmeier, Maj. Samuel G., Fort Oglethorpe, Ga.
Krukowski, Lt. Stanley M., 425 Taylor Ave., Collingswood, N. J.
McBride, Norman L., 1021 Davis St., Evanston, Ill.
Magens, Capt. Hans J., 619 S. Cedar, Little Rock, Ark.
Moses, Harold E., Ohio State University, Columbus, Ohio.
Mydland, Lt. Haldor T., 307 Metz Apt., 2009 Summit, Sioux City, Iowa.
Nielmeyer, Theodore J., 2121 Pico Blvd., Santa Monica, Calif.
Reese, Lt. William C., Earlville, N. Y.
Riser, Wayne H., Glenwood, Iowa.
Saunders, Lt. Charles M., Fort Missoula, Mont.
Seagers, Lt. William J., Apt. 9, 47 Cedar St., Binghamton, N. Y.
Seibert, Zen W., Crestline, Ohio.
Shipley, Lt. Wayne D., 226 Sixth Ave., Columbus, Ga.
Shoaff, Capt. Walter P., 217 Buena Vista, Paris, Ill.
Spring, Lt. Jacob E., 608 E. Missouri Ave., Saint Joseph, Mo.
Tierney, Lt. William F., 38 S. Hermitage Ave., Trenton, N. J.
Trum, Lt. Bernard F., Army Veterinary School, Army Medical Center, Washington, D. C.
Whitfield, Lt. John S., Fort Oglethorpe, Ga.
Wilder, Clifford W., Chatham, N. Y.
Wilke, David C., 7 Quincy Court, Pittsburg, Kan.
Willis, Lt. Robert L., Fort Oglethorpe, Ga.
Winston, James S., 9088 Santa Monica Blvd., West Hollywood, Calif.
Yost, Capt. Hursh R., Somerset, Ohio.

The amount which should accompany an application filed this month is $5.42, which covers membership fee and dues to January 1, 1937, including subscription to the JOURNAL. It is suggested that applications filed this month be accompanied by remittance for $10.42, the additional $5.00 being for the 1937 dues.

COMING VETERINARY MEETINGS

East Tennessee Veterinary Medical Society. White Surgical Supply Building, Knoxville, Tenn. December 5, 1936. Dr. Robert L. Hummer, Secretary, 312 W. Church Ave., Knoxville, Tenn.

Northeastern Indiana Veterinary Medical Association. Fort Wayne, Ind. December 8, 1936. Dr. Geo. L. Clark, Secretary, Columbia City, Ind.

Nebraska State Veterinary Medical Association. Lincoln, Neb. December 8-9, 1936. Dr. J. D. Sprague, Secretary, David City, Neb.

Western Michigan Veterinary Medical Association. Dr. J. Y. Veenstra's Small-Animal Hospital, Grand Rapids, Mich. December 10, 1936. Dr. Chas. H. Haasjes, Secretary, 728 S. State St., Shelby, Mich.

South Dakota Veterinary Medical Association. Cataract Hotel, Sioux Falls, S. Dak. December 10-11, 1936. Dr. Geo. E. Melody, Secretary, Gettysburg, S. Dak.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. December 15, 1936. Dr. C. C. Foulk, Secretary, 1103 E. 47th St., Kansas City, Mo.

Vermont Veterinary Medical Association. Montpelier Tavern, Montpelier, Vt. December 15, 1936. Dr. G. N. Welch, Secretary, 43 Union St., Northfield, Vt.

Massachusetts Veterinary Association. Hotel Westminster, Boston, Mass. December 16, 1936. Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. December 16, 1936. Dr. L. E. Pike, Secretary, 1220 Bennett Ave., Long Beach, Calif.

Delaware Veterinary Medical Association. Hob Tea Room, 9th and Market St., Wilmington, Del. December 22, 1936. Dr. Charles I. Hock, Secretary, 904 Jackson St., Wilmington, Del.

American Association for the Advancement of Science. Washington, D. C. December 28, 1936-January 2, 1937. Dr. Henry B. Ward, Secretary, Smithsonian Institution Bldg., Washington, D. C.

Keystone Veterinary Medical Association. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. December 30, 1936. Dr. M. W. Allam, Secretary, Media, Pa.

Southern California, Veterinary Hospital Association of. Los Angeles, Calif. January 5, 1937. Dr. L. B. Wolcott, Secretary, 1434 W. Slauson Ave., Los Angeles, Calif.

Pennsylvania, Conference for Veterinarians at University of. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. January 5-6, 1937. Dr. G. A. Dick, Dean, 39th St. and Woodland Ave., Philadelphia, Pa.

California State Veterinary Medical Association and University of California Veterinary Conference. University Farm, Davis, Calif. January 5-8, 1937. Dr. Chas. J. Parshall, Secretary, Brentwood, Calif.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. January 6, 1937. Dr. Milton R. Fisher, Secretary, 3678 Dover Pl., Saint Louis, Mo.

Houston Veterinary Association. Houston, Texas. January 7, 1937. Dr. D. B. Strickler, Secretary, 317 Federal Bldg., Houston, Texas.

Cornell University, Annual Conference for Veterinarians at. New York State Veterinary College, Ithaca, N. Y. January 7-8, 1937. Dr. W. A. Hagan, Dean, Cornell University, Ithaca, N. Y.

New Mexico Veterinary Medical Association. State College, N. M. January 8-9, 1937. Dr. T. I. Means, Secretary, Penn Road, Santa Fe, N. M.

Ak-Sar-Ben Veterinary Medical Association. Elks Building, Omaha, Neb. January 11, 1937. Dr. J. N. McIlnay, Secretary, 3251 Leavenworth St., Omaha, Neb.

Intermountain Livestock Sanitary Association. Ogden, Utah. January 11-13, 1937. Dr. D. E. Madsen, Secretary, Utah Experiment Station, Logan, Utah.

Oklahoma Veterinary Medical Association. Skirvin Hotel, Oklahoma City, Okla. January 11-12, 1937. Dr. C. H. Fauks, Secretary, 1719 S. W. 15th St., Oklahoma City, Okla.

Chicago Veterinary Medical Association. Palmer House, Chicago, Ill. January 12, 1937. Dr. O. Norling-Christensen, Secretary, 1904 W. North Ave., Chicago, Ill.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. January 13, 1937. Dr. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Kansas Veterinary Medical Association. Allis Hotel, Wichita, Kan. January 13-14, 1937. Dr. Chas. W. Bower, Secretary, 1128 Kansas Ave., Topeka, Kan.

New Jersey, Veterinary Medical Association of. Hotel Douglas, Newark, N. J. January 14-15, 1937. Dr. J. G. Hardenbergh, Secretary pro tem., c/o Walker-Gordon Laboratory Co., Plainsboro, N. J.

Ohio State Veterinary Medical Association. Deshler-Wallick Hotel, Columbus, Ohio. January 14-15, 1937. Dr. R. E. Rebrassier, Secretary, Ohio State University, Columbus, Ohio.

Wisconsin Veterinary Medical Association. Park Hotel, Madison, Wis. January 18-20, 1937. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

South Carolina Association of Veterinarians. Jefferson Hotel, Columbia, S. C. January 19, 1937. Dr. R. A. Mays, Secretary, 408-410 State Office Bldg., Columbia, S. C.

Indiana Veterinary Medical Association. Severin Hotel, Indianapolis, Ind. January 19-21, 1937. Dr. W. B. Craig, Secretary, 1420 N. Alabama St., Indianapolis, Ind.

Iowa Veterinary Medical Association. (Place not stated in notice.) January 19-21, 1937. Dr. C. J. Scott, Secretary, Knoxville, Iowa.

Texas, State Veterinary Medical Association of. Fort Worth, Texas. January 20-21, 1937. Dr. Dee Pearce, Secretary, Box 335, Leonard, Texas.

Colorado Veterinary Medical Association. Albany Hotel, Denver, Colo. January 21, 1937. Dr. B. R. McCrory, Secretary, Colorado State College, Fort Collins, Colo.

Minnesota State Veterinary Medical Society. Hotel Saint Paul, Saint Paul, Minn. January 21-22, 1937. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

Nevada State Veterinary Association. Reno, Nev. January 22, 1937. Dr. Warren B. Earl, Secretary, Box 1027, Reno, Nev.

Michigan State College Short Course for Veterinarians. Michigan State College, East Lansing, Mich. January 25-29, 1937. Dr. Ward Giltner, Dean, Michigan State College, East Lansing, Mich.

Missouri Veterinary Medical Association and Special Course for Graduate Veterinarians. University of Missouri, Columbia, Mo. January 26-28, 1937. Dr. C. L. Campbell, Secretary, 1817 Holmes St., Kansas City, Mo.

Mississippi State Veterinary Medical Association. Tupelo, Miss. January 28-29, 1937. Dr. E. H. Durr, Secretary, Clinton Blvd., Jackson, Miss.

STATE BOARD EXAMINATIONS

California Board of Examiners in Veterinary Medicine. University Farm, Davis, Calif. January 4-6, 1937. Dr. Nelson E. Clemens, Secretary, 183 Castro St., Hayward, Calif.

Connecticut State Board of Veterinary Registration and Examination. State Office Building, Hartford, Conn. January 5, 1937. Dr. Geo. E. Corwin, Secretary, State Office Bldg., Hartford, Conn.

Iowa Veterinary Medical Examining Board. State Capitol, Des Moines, Iowa. January 18-19, 1937. All applications must be in the office of the Division of Animal Industry not later than 8:00 A. M. on January 18. Further information may be obtained from the Secretary. Dr. H. A. Seidell, Secretary, State Capitol, Des Moines, Iowa.

SOME PHASES OF SWINE PRACTICE*

By JOHN B. BRYANT, *Mount Vernon, Iowa*

About a quarter of a century ago, equine practice was on the wane and the diseases of food-producing animals were in the ascendancy. The lowly swine had acquired versatility, having proved their ability to entertain many other ailments than hog cholera. Since that time, learned works have been published on swine diseases. With the dissemination of correct knowledge, swine practice has assumed worthy proportions, acquired many refinements, and encountered many serious problems. This paper is an attempt to portray some phases of swine practice as encountered and as handled by a general practitioner, aided and abetted by research workers, complicated by legislative acts, remedy peddlers, existing field conditions, and the vagaries of owners.

SWINE PRODUCTION

Selection of parent stock and breeding operations: Select a vigorous, rugged sire, and a maternal type of dam, deep in the body, broad in the pelvis, with about one dozen prominent teats. Some breeders give preference to sires that have numerous and prominent teats. The gilt may be bred at nine months. According to swine authorities, the rule is that nursing sows do not conceive until three to five days following weaning. I observe exceptions to this rule, providing the boar runs at large with the nursing sows. Many breeders practice rather heavy feeding, termed "flushing," at breeding time. Pen breeding is advised, allowing the boar two services daily. According to Kinsley, pen-bred sows usually average two to four times more pigs per farrow than do pasture-bred sows.

The pregnant sow: Guard the pregnant sow's physical condition and avoid swine flu, mange and lice. Feed her a balanced ration, consisting of a variety of feeds, to which a mineral is added. Corn and water alone do not produce good litters. Factors most commonly neglected in the pregnant-sow ration are minerals, protein, hay, and an abundance of fresh water. Another essential is exercise. As the sow approaches parturition, her exercise will be reduced, hence the ration should be reduced. When the sow is confined in the parturition pen, the ration should be about 50 per cent of the full allowance.

The farrowing sow: Peace and contentment should reign in the maternity pen. A full dose of Epsom salt usually allays

*Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

fever and quiets a nervous sow. The first 24 hours following parturition, the sow should lie still except for the time she is up to relieve her bowels and bladder and to get a drink of fresh water. The second day, she may have a light feed of oats and bran. This may be increased gradually and supplemented day by day until the tenth day, when the full milk-producing ration may be given, providing all is going well with the litter. The idea during the first ten days is to satisfy the sow with bulky, non-milk-producing feeds which assist in abating the fever of farrowing and the local fever in the freshened udders, so that the milk will not cause scouring in the pigs. A scant supply of milk also prevents overloading of the baby pigs and the consequent dietary scours.

Swine dystocia: The first step is to cleanse the sow's external genitals and the hands and to lubricate them. I use castile soap. Next, I inject water into the vagina, and pass the bar of soap, grasped with the vulsellum forceps, up and down the vaginal canal to lubricate it. Then I explore the vaginal and pelvic accommodations, to ascertain whether multiple delivery can be accomplished without irreparable damage to the parts.

Vaginal delivery: The instruments I use are a No. 9 wire hook and a Day's pig-snare. The lower jaw is grasped, the wire hook passed down the arm and fastened in the bifurcation of the jaw, and delivery attempted. If the required traction threatens to fracture the fused bones, I then pass the snare over the hook to the pig's nose. The hook serves to fix the pig's head while the snare is passed over the poll and closed under the lower jaw. Usually this requires patient, skillful manipulation. Now carefully pull on the snare with one hand, and guide the pig's head and slip back the vaginal walls with the other hand. If it is a posterior presentation, the snare may be applied to one or both hind legs. Always in vaginal deliveries, the question arises as to whether delivery is complete. External palpation offers me some help. With one hand under the sow, in the flank region, and the other hand in a similar position, on the upper side of the sow, one may frequently palpate pigs if they are present. If no pigs can be detected, and the delivered pigs are the average of the other litters in the farrowing house, it is my guess that the sow is done and my guess seldom is wrong.

Cesarean section: In cases of uterine inertia, I have had little help from pituitrin, ergot, or other labor-stimulating agents. In these cases, and in cases of insufficient pelvic accommodations, I resort to cesarean section. Neglected parturition cases and those cases that have been subjected to prolonged manipulation are

held to be unfavorable cesarean subjects. I will not take time to describe the operation, as it will be demonstrated at the clinic.

Phases of Baby-Pig Practice

Pig scours: Usually this condition is due to errors in feeding the sow or to filth on the sow's udders. Correct these errors, cleanse the sow's alimentary canal with Epsom salt and administer sulfocarbolates compound two or three times daily in dampened oats, slop or drinking water. Another treatment is a saline laxative followed by an alkaline compound, given to the sow, on soaked oats.

Necrobacillosis of baby pigs: I do not favor clipping the black teeth, as it opens avenues for infection. The sore-mouth form may be treated by curetment and tincture of iodine. The lesions on the side of the face are easily overlooked. They may be treated with a wound oil applied with an oil-can. I recommend examination of all baby pigs when they are two to three days old and the antiseptic oil treatment of any and all skin abrasions, and also of the navel. Repeat every second or third day, or as needed, until the pigs are three weeks old.

Baby-pig anemia: This is prevented by getting the pigs on the ground or by carrying sods into the farrowing house. Another method is to paint the sow's udders with an iron and copper compound. Diagnose anemia with the hemoglobin scale. These pigs are plump and dropsical. The tissues are anemic. The heart muscles are atonic.

External parasites: Mange and lice greatly damage sucklings and must not be overlooked. Treat by dipping in lime and sulfur solution or crude oil, properly prepared and used.

The weanling pig: Preparation for weaning should begin at about four weeks of age. Provide a creep wherein the little fellows are permitted shelled corn and hulled, rolled, or whole oats of good quality. At six weeks, a slop may be provided. At seven or eight weeks, administer anti-hog cholera serum and virus and remove the sows at nine or ten weeks, or wean at eight weeks and vaccinate at ten weeks. I do not endorse tankage or any material change in the ration during the period of 17 to 21 days following the serum-virus treatment.

Phases of Shote Practice

The acid test in swine practice comes with the handling of the variety of ailments found in hogs of the shote age. The art of swine practice requires a knowledge of swine habits in health and disease, a knowledge of postmortem technic, pathology,

immunology, therapeutics, patient, keen observation of the hogs, and diplomatic dealings with owners.

Hog cholera: While listening to a lecture by the late Dean Stange, a note was taken, as follows:

> To establish an infectious disease, it is necessary to have the infectious agent, the path of the infection, and the susceptible host. In the control of the disease, one of these factors must be eliminated.

We all know the famous trio who discovered the causative factor of hog cholera and recognized the futility of destroying it. They found that the path of the infection could not be controlled. With the production of anti-hog cholera serum and hog cholera virus, they opened the way for eliminating susceptible hosts. Few of our clients understand this chain of factors, and misconceptions abound when hog cholera is enzoötic. Each fall, when shotes are started on new corn, we witness outbreaks of hog cholera and many owners are convinced that new corn is the causative factor. We should carefully advise such owners that hog cholera is a germ disease, that new corn is simply a contributing factor, and that any sudden change of feed lowers resistance, thereby rendering hogs quite susceptible to hog cholera.

Each year, during threshing and silo-filling, some farm in one of the rings may be infected with hog cholera. Neighbors in the ring become apprehensive concerning the transmission of the infection. Dr. C. N. McBryde's tracking experiments were negative, yet it is professionally sound to advise that straw be spread down and saturated with a strong disinfectant solution, over which teams and wagons and men afoot pass before leaving the infected premises. Doctor McBryde did transmit cholera with flies. Flies will follow horses and ride in automobiles away from cholera-infected farms. Therefore, farmers having cholera-sick hogs should attempt to control these fly hazards by keeping their hogs confined in darkened quarters. Threshing should be done in the field, avoiding the hog lots. Teams leaving infected farms should be sprayed with a fly repellent and flies should be shooed out of automobiles.

Frequently, an owner of cholera-sick hogs asks the involved question, "Where did I get this cholera?" I dislike that question. How do you answer it, or is it the part of diplomacy to evade it? The germs are too small to be seen. Even if they were as large as flies, it might be hard to trace them, if they were harbored under some old building or escaped from some forgotten cholera grave, or alighted from some rendering truck containing hogs

dead of cholera. Again, many owners of cholera-afflicted droves carefully conceal the fact but not the decaying carcasses. The lay distribution of hog cholera virus unlooses these death-dealing germs to the high winds. Hogs are vaccinated, loaded and shipped from central markets to new surroundings, new management and changed feed, to undergo the serum-virus reaction, and mayhap establish new centers of cholera. The community sales barn, that mecca of live stock infections, sends the seeds of swine diseases into the small-farm drove as well as the large one.

"Where did I get this cholera?" If you, brother practitioner, raise your finger, it is very likely to fall on some permit-holder, or some careless owner who would resent the assertion; or it might be some hog dealer who is a client of yours and whose large vaccination check is in your pocket. Many such owners are sensitive; they hold that they are paying you to control their hog diseases and not to advertise them to neighbors or prospective customers. Have you had this experience? I have, and yet your clientele has a right to expect you to interest yourself in protecting the community against devastating epizoötics. This can be promoted through publicity of the area type. The Eastern Iowa Veterinary Association, Inc., for eleven years, has issued weekly bulletins to the press of that district, designating by towns the location and the extent of menacing outbreaks of hog cholera and other infectious diseases. This service is of unquestioned economic value to hog-owners who appreciate the warning and commend coöperating practitioners, as my Eastern Iowa colleagues will testify.

The slow incidence of the usual case of hog cholera presents a practical problem. Many owners attach slight significance to one or two shotes that are off feed and do not call for help until several are sick, or dead. Usually, by this time, the disease has reached alarming proportions and it behooves the practitioner to convince the owner of the seriousness of the case. Use a thermometer diligently. Be sure to test some that have just passed the incubation period. They are a little slow at feed, a little stilty in their gait, and their sides drop a little but they have escaped the owner's attention. High temperatures in several of these are convincing. I then explain that, in general, the incubation period is approximately five days. The disease is then established, as evidenced by a rise in temperature. However, such affected swine will follow their mates and disseminate the disease for approximately another five days before going off feed and becoming visibly sick. I want these owners to know

that hog cholera has been present in their droves for approximately ten days and I want them prepared for the losses that will surely ensue.

A question sometimes asked is when and when not to use virus. It is quite generally the practice to use virus whenever serum is used. Serum alone affords an uncertain and variable protection. We encounter droves on cholera-infected premises where sows have just farrowed, are farrowing, or will farrow in a few hours or a few days. Is there an indication here for serum alone? No. Treat all sows and pigs with serum and virus, inject the baby pigs with about 16 to 20 cc of serum, intraperitoneally, and 1 cc of virus in the loose tissue of the lower flank region, using a one-inch 20-gauge needle inserted to the hub, painstakingly pinch and hold the puncture to prevent leakage, and finally apply the antiseptic. These baby pigs are subjects for tedious administration. Hold these babies separate until the convulsions occasioned by the serum have subsided, to avoid trampling by the sow. Now, what shall we do with the baby pigs farrowed to a sow while in the serum-virus reaction period? I recommend serum-virus treatment at two to three days old. You will not all agree that this is necessary. Emergency-treated baby pigs are to be vaccinated again at the weaning period.

Baby-pig vaccination: On September 20, 1935, 36 suckling pigs, 20 to 25 pounds in weight, were vaccinated. Two months later, on the same farm, 42 suckling pigs weighing 20 to 25 pounds were vaccinated. All of the pigs were on immune mothers. The first group was finished and marketed without loss. On June 20, 1936, losses started in the second group. After five head had died, now 125-pound shotes, the balance were retreated and losses were definitely terminated. Baby-pig vaccination is not safe in my territory.

Suggestions for administration: I use intraperitoneal administration in all pigs that can be held up by the hind legs or laid in the trough. Shotes that are too heavy for this method of restraint may be thrown on the floor by two men, one grasping a hind leg and the other the front leg on the same side. The man holding the front leg places a foot on the jowl of the head, and the man holding the hind leg places a foot on the under hind leg. The axillary and inguinal spaces are thus exposed for injection. The site is treated with a skin disinfectant that is also a stain, which serves for identification should occasion arise while vaccinating. When using intramuscular injections of serum, I prefer to inject not more than 20 cc at one place in swine up to 100 pounds and 30 cc in heavier hogs. When

vaccinating pregnant sows, I have them carefully crowded in a corner with a hurdle and caught about the snout, with a rope which is snubbed to a convenient post. I then inject back of the ears and forelegs. I never allow the throwing of a pregnant sow. I use a Shikles serum bag and a 40-cc syringe for the virus, one syringe in each hand. When applying the disinfectant from a self-fashioned dauber bottle, the two syringes are held in one hand. A carpenter's apron provides a pocket for the disinfectant bottle.

A vaccination hazard: In 1931, I was called to vaccinate 75 pigs confined on a feeding floor, just off of which was a filthy mud hole, suggesting lurking infections. I mentioned this hazard and required a thorough flushing of the floor. I also ordered the vaccinated pigs held on the cleansed floor overnight, which was calculated to permit closure of the puncture wounds. The next day, eight pigs died with typical gas infection. This single experience hardly qualifies me to give definite advice on this serious point. It is recommended that equal parts of gasoline and tincture of iodine be applied to injection sites as a resolvent and disinfectant and that collodion be used as a sealing agent for the needle wounds.

There is a matter I deem of great importance which devolves upon the practitioner. We demand a potent, sterile serum and a virulent and uncontaminated virus. It is our duty to guard these quality products until they are delivered into the hog. The virus stock must be stored at 50° F. A cool cellar will suffice for the serum. The virus must be iced when taken to the field in hot weather. I had a tinner make for me an inexpensive and attractive virus box. A wide-mouthed thermos jug may be iced and is a satisfactory virus receptacle.

Swine dysentery: This is a very serious disease as many of you may testify. It is characterized by bloody diarrhea, varying degrees of inappetance, and mortality, which probably depends upon the virulence of the infection and the resistance of the affected swine. Many terms dignify this disease, as swine dysentery, infectious hemorrhagic enteritis, bloody dysentery, and others. This disease is prevalent and causes terrific losses on many farms where hogs are imported for feeding purposes and to follow cattle in the feed lot. But cattle droppings and imported hogs are not prerequisites of this disease. I have seen it suddenly appear in native farm-raised droves of hogs.

Someone has suggested that it might be rational to stop research and devote more time to the application of human knowledge. I feel that research workers have done little toward

charting a course for the practitioner who encounters swine dysentery. True, experiments have been conducted on transmission, incubation periods have had some attention, the causative agent or agents have been found elusive, and the studies of the lesions have disclosed but little of benefit to the practitioner. While research workers are pondering this disease or neglecting its intensive study, the malady is exacting an enormous toll and practitioners are forced to experiment with ways and means for retaining some degree of owner confidence. The omnipresent hog remedy peddler boldly enters the field, foisting his concoctions on distracted owners. The whole serious mess is an eloquent appeal for research work on this and related enteric diseases of swine. The Section on Research of this great association of ours, now in convention, has no report of this increasing menace to the swine-raising and cattle-feeding industries. A very complete treatise on swine dysentery[1] was read before the United States Live Stock Sanitary Association by Dr. R. M. Hofferd, last year. His field experiences prompted the following extracts:

> In the field, we continue to hope that the etiology will soon be cleared up in order that more specific control measures may be established to obliterate this increasing menace to the swine-raising and cattle-feeding industries. * * * The chief measure effective in controlling this disease has been sanitation. * * * The treatment of droves affected with this disease offers a very grave problem.

Control and treatment: Some prefer to confine the hogs in a dry lot. Others prefer a fresh pasture and some place the hogs on a cement floor, which is flushed off several times daily. I prefer a clean alfalfa or clover pasture. At this time of year (August), a corn field is a very fine place. Control of infection intake is paramount. On some farms, sanitation is simple, while it is complicated indeed on other farms, usually those where commercial feeding of cattle and hogs is carried on, or in the winter, when all available hog quarters are in use. Each case is a problem in itself.

I have contacted several of my colleagues and present herewith some of the drugs used in attempting to treat swine dysentery:

Formalin, bicarbonate of soda, Cooper's Kerol, castor oil, alkaline compounds, sulfates of copper, iron and magnesium, pyoktanin blue, arsenous acid, gentian violet, and lactic acid-bearing feeds. Biologics have been used on some cases with complete success and, in other cases, with apparently dismal failure. Do not mistake this disease for hog cholera, especially

if the affected swine have been vaccinated by yourself, and more especially if vaccinated by a neighboring practitioner. These hogs are more lively, scour earlier, carry a lower temperature and come down in larger numbers than do hogs affected with cholera.

Necrotic enteritis: This condition is a filth-borne disease. We all have our enteritis farms, where the young pigs do well until about the time they are eating corn. The first symptom is loose bowels, then varying degrees of scouring. Appetites remain good in the chronic form and the pigs gradually shrink away to mere skeletons. The very acute type of this disease closely resembles swine dysentery. Treatment consists in moving the pigs to clean ground and feeding alkalinized soaked oats and milk feeds. Eliminate corn and especially tankage from the ration.

Swine-pox: In October, 1935, I observed two droves of hogs, separated by a highway, and attended by the same caretaker. One drove consisted of suckling pigs and the other drove of shotes. They were not cholera-immune. Many of the pigs were extensively affected with pustules and with temperatures up to 106° F. The diagnosis was swine-pox. Treatment used in this case was spraying with a light oil to control lice and mange which are factors in swine-pox. This condition ran its course in about ten days. Individual pigs ran the course in three to six days. There was about a 40 per cent loss in the suckling pigs and those that survived were slow to gain. The drove of shotes withstood the affliction with no loss in numbers and slight loss of condition. This is the only case I have seen that caused heavy loss. Swine-pox is frequently seen as a slight affection on the bellies of occasional pigs presented for vaccination. Avoid the use of dips in these cases, owing to the high susceptibility of pigs to cresylic acid poisoning, due to the open pock wounds.

Sun scald: A condition that may confuse the uninitiated is what I term "sun scald." Any forage that has grown tall will carry the water from dews or rains and drench the backs of hogs as they graze. If such wet hogs are exposed to the direct hot rays of the sun, they are apt to become scalded. The backs of Chester Whites are very susceptible, also the white belt of Hampshires. The scalded areas form scabs which crack and the resulting fissures may bleed and even suppurate. Such hogs run temperatures, feed sparingly, and assume attitudes closely resembling the clinical picture of hog cholera. Another symptom is severe flinching or even squealing of affected hogs when

crowded by mates while feeding. Treatment consists of removal from forage while wet and spraying with light antiseptic oil.

Hog-ring infection: Rings set too deeply in the snouts of hogs may cause infection, with resulting high temperatures and inappetance. Do not mistake this for cholera. The treatment is removal of the rings and applications of tincture of iodine.

Surgical Interventions

I will describe ordinary castration, scrotal hernia, and ridgling castration.

The surgical landmark of these operations is the external inguinal ring, located in the middle inguinal region. The regional anatomy includes the scrotal canal extending from the ring to the scrotum, situated a short distance below the anus. This canal contains the tunica vaginalis, a pouch of the peritoneum, which encases the spermatic cord and the testicle. The tunic is attached loosely in the scrotum. I want my subjects held up by the hind legs or stretched in the trough head down, as for vaccination. I grasp the skin at the posterior third of the scrotal canal and, with a hook knife, incisions are made on each side and parallel with the median raphé, and carried down into the scrotal canal but not through the tunic. The tunic is now pulled loose from its scrotal attachments and removed with its contained testicle by a few scraping strokes toward the ring. It is well to observe the inguinal ring. If it is large and intestinal prolapse threatens, reduce the ring with a suture. If abscess formation is a later development in the scrotal cavity, a simple incision will evacuate the fluid pus.

Scrotal hernia: The incision is over the inguinal ring parallel with the median line and is carried into the scrotal canal. The tunic and contents are exposed down to the ring. The sac is then twisted, which forces the intestines back into the peritoneal cavity. A hemostat is now placed near the ring and a figure-eight ligature, which passes through the cord twice, is firmly applied. The tunic and testicle are then removed with the scissors above the hemostat. The ring may be reduced with a suture, if desired as an extra precaution. No further suturing is necessary.

Ridgling castration: The incision starts over the inguinal ring and is carried anteriorly and medially, in direction, and down to the abdominal muscles; these are divided by blunt dissection down to the peritoneum, which is picked up and incised to admit one finger and a canine spaying-hook. The testicle is then located with the finger and brought through the incision with

the hook and removed. The peritoneum may be closed with a gut suture. No further sutures are needed, as the parts will close the surgical tract, when the pig comes to a normal position.

Reference

[1]Hofferd, R. M.: Swine dysentery in Iowa from a field standpoint. Jour. A.V.M.A., lxxxviii (1936), n. s. 41 (3), pp. 299-310.

Discussion

Dr. C. C. Hastings: Dr. Bryant, you spoke of giving Epsom salt for pregnant sows. What is your dosage and what is your method of administration?

Dr. Bryant: It depends somewhat on the size of the sow. I give a big sow a couple of rounding tablespoonfuls, all you can get on the tablespoon and a little more. Snub them with a rope and give it to them with a dose syringe.

Dr. T. H. Ferguson: There was one statement that I was very glad to hear Dr. Bryant make, and that was about the administration of serum and virus to hogs sick with cholera. My experience in treating hogs has been rather limited, although I have treated some very valuable herds. It has always been my practice in treating sick herds not to depend on the yard infection of the virus but treat all of them with serum and virus at the same time.

In any case, whether you use serum or virus, you are going to have some deaths, but if you use serum and virus instead of serum alone, after the smoke clears away, what hogs are left will go on and do well and produce a good profit to the owner. If you use serum alone and depend on your yard infection, you will have a lot of runty, chronic cases of cholera and these pigs will never do well. That has been my experience, and I was glad to hear the doctor bring out that particular point.

Dr. W. C. Bateman: I should like to ask Dr. Bryant if he has had any experience with this so-called three-day pig sickness that we have been bothered with in California. The pigs die within two or three days.

Dr. Bryant: I do not believe I have. I have not heard it called that. I wish you would come up and tell us about it.

Dr. Bateman: The sows seem to farrow normally but the pigs are weak and develop scours and then all will be dead in three days. Some of the sows will have nice pigs and everything will be O. K., and maybe the next sow will lose all of hers. It goes through the whole herd that way. Dr. McBryde was out there a few years ago, doing some experimental work, but I have never heard much about it.

Dr. Hastings: Does that occur in gilts or is that a disease of aged sows?

Dr. Bateman: Both old sows and gilts.

Dr. Hastings: Is it not a fact that that occurs mostly in gilts?

Dr. Bateman: Yes. However, it goes through all of them.

Dr. Hastings: That is very prevalent in purebred herds, especially in herds where the gilts have gone through a case of swine flu before they were bred. The gilts that have the flu will usually produce pigs that are very weak. They are commonly known among the farmers as "squealers." They do not look exactly right; they have silky hair, rather thin, white, light-colored skin, and they run around the pen. They leave the sow, and you find them lying over in one corner of the pen dead 24 or 48 hours later.

In central Illinois we have a great deal of it, especially among our purebred herds. Ordinarily, as I said a moment ago, it is confined to

the gilts, especially if the gilts have been bred to a young boar. Sometimes we find it in the old sows, if they have been bred to a young boar that has gone through a case of swine flu sometime before he served those sows. That ordinarily is a complication or a sequel of swine flu, or sometimes a dietary trouble. Sows that are under-nourished and have had very little exercise during their pregnant period will produce weak, squealy pigs, but the strong, healthy, vigorous pig is the pig that the farmer makes his money on, the product of an aged sow and an aged boar mated. The weak, little fellows are ordinarily the product of breeding stock that has been bred prematurely.

BUREAU TRANSFERS

DR. RAY O. PORTER (K. C. V. C. '09), from Clarksville, Ark., to Sacramento, Calif., on tuberculosis eradication.

DR. GROVER C. GULICK (K. C. V. C. '16), from Kansas City, Kan., to Marshalltown, Iowa, on meat inspection.

DR. ABRAHAM A. KRITT (O. S. U. '20), from Manchester, N. H., to Albany, Ga., in charge of meat inspection.

DR. WM. J. SCANLON (K. S. C. '15), from Sioux Falls, S. Dak., to Kansas City, Kan., on meat inspection.

DR. GEORGE M. OTIS (Chi. '03), from Milwaukee, Wis., to Chicago, Ill., on meat inspection.

DR. HARPER H. SHEARER (Iowa '20), from Chicago, Ill., to Albany, Ga., on meat inspection.

DR. CHAS. P. BRADY (K. C. V. C. '13), from Fort Worth, Texas, to Denver, Colo., on tuberculosis eradication.

DR. JAMES A. JOHNSON (O. S. U. '34), from Marshalltown, Iowa, to Kansas City, Kan., on virus-serum control.

DR. IVAN L. BARSTOW (K. C. V. C. '10), from Moscow, Idaho, to Denver, Colo., on meat inspection.

DR. FRANK A. HENNEY (K. C. V. C. '18), from Saint Paul, Minn., to Denver, Colo., on tuberculosis eradication.

DR. WM. B. CHAPMAN (St. Jos. '22), from Boston, Mass., to South Saint Joseph, Mo., on meat inspection.

DR. DAVID J. RYAN (K. C. V. C. '11), from Austin, Minn., to Fort Worth, Texas, on meat inspection.

DR. WILBUR R. KIDWELL (O. S. U. '19), from Sioux Falls, S. Dak., to Chicago, Ill., as assistant inspector-in-charge of meat inspection.

DR. WILLIAM C. GLOCKNER (U. P. '20), from Jackson, Miss., to Baton Rouge, La., on tuberculosis eradication.

DR. MYRON D. MOSES (Chi. '16), from Chicago, Ill., to Evansville, Ind., on meat inspection.

DR. LAURENCE D. BOSTON (Colo. '32), from Evansville, Ind., to Wichita, Kan., on meat inspection.

DR. HUGH J. CLARY (St. Jos. '20), from Winona, Minn., to South Saint Paul, Minn., on meat inspection.

DR. EDWARD E. MAAS (K. C. V. C. '17), from Fort Worth, Texas, to San Juan, P. R., on tuberculosis eradication.

DR. GUY F. OVERHULSE (Wash. '11), from Omaha, Neb., to Spokane, Wash., on meat inspection.

DR. NORTON A. ORR (Colo. '33), from Phoenix, Ariz., to Sioux Falls. S. Dak., on meat inspection.

CRYSTAL-VIOLET VACCINE FOR THE PREVENTION OF HOG CHOLERA: PROGRESS REPORT*

By C. N. McBryde *and* C. G. Cole
Field Station, Biochemic Division
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Introduction

Not content with his great achievement of developing a successful antiserum for the prevention of hog cholera, the late Dr. M. Dorset sought to develop a vaccine which would afford a cheaper and safer method of immunization against this devastating disease. He began the attempt at the Field Station of the Biochemic Division of the U. S. Bureau of Animal Industry, at Ames, Iowa, some 15 years ago. Vaccines were prepared from the blood and tissues of cholera-infected pigs by the use of various attenuating agents, such as formalin, ammonia, chloroform, glycerin and phenol. Much time and work were devoted to the preparation and testing of such vaccines. These experiments were not carried on continuously, but frequently an interval of one or more years elapsed after one attenuating agent was tried and found to be unsatisfactory before another method was taken up. Sufficient work was done with each of the attenuating agents, however, before it was abandoned. All the attenuating agents just mentioned sometimes yielded effective vaccines, but the results obtained were never uniform and consistent, when repeated lots of vaccine were prepared and tested.

During the last years of his life, when it seemed well-nigh useless to hope for success, Dr. Dorset decided to try crystal violet as an attenuating agent. Before he was stricken by the brief illness which culminated in his death in July, 1935, the results obtained with this dye, especially in conjunction with a small amount of phenol, had become so promising that he was keenly gratified. If crystal-violet vaccine fulfills the promise it has thus far given in our experimental work at Ames and becomes established as a successful method of immunization against hog cholera, it will prove a fitting climax to a brilliant scientific career.

Preparation and Testing of the Vaccines

In order to avoid unnecessary variables, practically all the vaccines produced at the Ames station have been made from

*Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

one lot of dye, which was sent to Ames by Dr. Dorset, bearing the label: "Crystal-Violet, Extra Pure, DuPont, Lot 42607, September 17, 1931."

The vaccine is prepared from the defibrinated blood of virus pigs, which are killed usually on the seventh or eighth day after injection. As a rule, the mixed blood of two virus pigs was used in the preparation of each lot of vaccine. Particular attention was given to the temperature records, clinical symptoms and postmortem lesions of these pigs, and care was taken to select pigs which would correspond in these respects with pigs used in the production of simultaneous virus at commercial establishments. Cultures were always taken from the tissues of these pigs at autopsy, to be sure that they were free from *Salmonella suipestifer* or other secondary infections.

At first, vaccines were prepared from the blood of cholera-infected pigs with crystal violet alone. Later, with a view to inhibiting bacterial growth and thus enhancing the keeping qualities of the vaccine, small amounts of phenol and orthocresol were added. Still later, the phenol and cresol, both of which tend to cause gelatinization, were omitted and the blood was rendered more alkaline by the addition of a small proportion of disodium phosphate. After the addition of the crystal violet and preservatives to the virus blood, the mixtures were placed in an incubator at 37.5° C. and held there for two weeks, as previous experimentation had shown this period of incubation to be required for the proper attenuation of the virus. Upon removal from the incubator, the vaccines were checked bacteriologically by making agar plates after suitable dilution to overcome the inhibiting action of the crystal violet.

In testing the antigenic or protective properties of the vaccines, susceptible pigs weighing from 40 to 90 pounds were used. Care was taken in the selection of these pigs to see that they were in good thrifty condition and also that they had been farrowed by non-immune sows. At the Ames station, the supplying of pigs for experimental purposes is restricted to farmers who do not practice immunization against hog cholera, for it has been found that pigs from immune sows may retain some degree of immunity after reaching the size required for experimental purposes. The susceptibility of each lot of pigs obtained for experimental purposes is determined as soon as possible by the injection of several of the pigs with active virus. The vaccines were tested in 5-cc and 10-cc doses and a comparison was made of the subcutaneous and intraperitoneal methods of administration. In administering the vaccines subcutaneously,

a single injection was made in the loose tissue of the inner side of the thigh.

It was found by experimentation that an interval of between two and three weeks is required for the establishment of immunity following vaccine treatment. The test pigs, therefore, were kept under close, daily observation for three weeks after receiving vaccine treatment and were carefully watched at feeding time. During this interval no temperatures were taken, as a rule, in order to prevent possible exposure through handling. In the case of two tests, however, the temperatures of the vaccine-treated pigs were taken with a view to determining whether any reaction follows vaccination. No significant temperature reaction was observed after the vaccine treatment and the animals did not show inappetence.

Three weeks after receiving vaccine treatment, the immunity of the animals was tested by the subcutaneous injection of 1 cc of virus of known virulence, which constitutes a much more severe exposure than would occur under field conditions.

VACCINES PREPARED WITH CRYSTAL VIOLET ALONE

These vaccines were prepared from the blood of cholera-infected pigs by the addition of five parts of a 1 per cent solution of crystal violet to 100 parts of defibrinated blood. Eight lots of vaccine prepared in this manner were tested on 26 pigs. All these pigs remained well following vaccine treatment. When exposed by virus injection, three weeks after vaccine treatment, 21 remained well and five showed a slight reaction, but quickly returned to normal. All the pigs in these tests may be regarded as having been adequately protected against hog cholera by the vaccine treatment and the protection afforded this group of pigs may be considered as 100 per cent or perfect.

Occasionally, the blood of virus pigs is found to contain *S. suipestifer* or *Escherichia coli*. Such bacterial contamination of the blood by secondary invaders is not uncommon in pigs which die from hog cholera, but rarely occurs in pigs killed for virus at the Ames station. Nevertheless, it does occur from time to time in pigs killed for virus on the seventh or eighth day. During the fiscal year ended June 30, 1936, 75 virus pigs were killed for the preparation of crystal-violet vaccines and cultures were made from the tissues of all of these pigs. *S. suipestifer* was obtained from the tissues of one pig killed on the seventh day after injection and from two others killed on the eighth day after injection. Inasmuch as plain crystal

violet is not strongly germicidal for the Gram-negative organisms of the colon-typhoid group, these contaminants can multiply during the incubation period in the case of plain crystal-violet vaccines. It became apparent that the product should be germicidally fortified.

Crystal-Violet Vaccines Containing Phenol

With a view to increasing bactericidal action, and thus enhancing the keeping qualities of the vaccines, experimental lots of vaccine were prepared containing 0.1 per cent, 0.2 per cent, and 0.5 per cent phenol. Because of the marked gelatinizing effect of even small amounts of phenol on defibrinated blood, it was decided to use only 0.1 per cent as an adjuvant. The majority of the crystal-violet vaccines which have been prepared have contained this percentage of phenol and these vaccines will be referred to as "regular vaccines." The method of preparation consists in adding one part of a one per cent solution of phenol to nine parts of defibrinated blood and then adding five parts of a one per cent crystal-violet solution to 100 parts of the previously phenolized blood. This method of preparation was furnished by the Bureau to serum producers and biological houses in December, 1935, with permission to prepare experimental lots of vaccine under government supervision. Since January 1, 1935, 42 lots of regular vaccine have been prepared at the Ames station. The great majority of these vaccines were found to be sterile at the end of the incubation period. Now and then the agar plates revealed an occasional spreader. In three instances the agar plates were found to be overgrown with *S. suipestifer* and in one instance with *E. coli*, and these vaccines were discarded.

The regular vaccines were tested on 187 station pigs and 20 farm pigs, or a total of 207 pigs. All these pigs remained well following vaccine treatment and all were later exposed to hog cholera by virus injection: One hundred fifty-five were exposed at the end of three weeks, four at the end of four weeks and 48 after an interval of from two to six months. The results of the exposure or immunity tests were as follows:

Remained well after exposure	184
Slight reaction, with rapid recovery	18
Severe reaction, with good recovery	3
Severe reaction, with poor recovery	1
Died	1
Total number of pigs treated	207

It would seem proper to regard the 18 pigs which showed only a slight reaction followed by a rapid return to normal as having been adequately protected, for the reaction was often so slight that it might have passed unnoticed, had the animals been treated on a farm. If these 18 pigs be combined with the 184 pigs which remained normal after vaccination, there were, out of 207 pigs, 202 which were successfully immunized against hog cholera by the vaccine treatment, or 97.6 per cent of the pigs treated in this group. It would not seem fair to regard the pigs which recovered after severe reactions as having been adequately protected, in spite of the fact that they made good recoveries, for a farmer would hardly regard these pigs as having been successfully immunized. The possibility that such pigs may have sickened as a result of some secondary infection rather than from the virus injection should be taken into account, however.

It may be noted in passing that three of the pigs which showed severe reactions were treated with one lot of vaccine, which was apparently lower in antigenic value than the others, but all of these pigs made good recoveries nevertheless. The pig which made a poor recovery after a severe reaction exhibited symptoms of pneumonia, rather than those of hog cholera, and probably was suffering from a secondary lung infection. The autopsy on the one pig that died in this group revealed a roundworm blocking the gall-duct and lesions other than those characteristic of hog cholera. If these facts be taken into account, the protection afforded by this group of vaccines was well-nigh perfect. However, the product was obviously deficient in bactericidal power.

CRYSTAL-VIOLET VACCINES CONTAINING ORTHOCRESOL

Experiments conducted in the laboratories of the Biochemic Division at Washington indicated that mixtures of orthocresol and crystal violet were strongly germicidal for *E. coli*. Therefore, vaccines were prepared by first adding ten parts of a 2 per cent solution of orthocresol to 90 parts of defibrinated blood and then adding to this mixture five parts of a 1 per cent solution of crystal violet. These vaccines, like the others, were incubated for two weeks to accomplish the proper attenuation of the virus. When checked bacteriologically at the end of the incubation period, these vaccines were found to be sterile. Fourteen lots have been prepared and tests have been completed on eight of these lots. Sixteen pigs were used in these tests. All the pigs remained well after vaccine treatment and were exposed

to hog cholera by virus injection at the end of three weeks. Fifteen of these pigs remained normal after virus injection, while one showed a slight reaction, followed by a rapid return to normal. Therefore, in a limited number of tests (eight lots tested on 16 pigs), these vaccines have given 100 per cent protection. In spite of the excellent results obtained with these vaccines, they would not constitute a desirable commercial product because of their physical condition. The addition of the orthocresol solution to defibrinated blood causes a marked gelatinization and the formation of a dark, gelatinous mass, which settles to the bottom of the glass containers as a thick jelly and adheres to the sides of the bottles in dark streaks. The gelatinized material at the bottom of the bottles breaks up readily, however, upon shaking.

Crystal-Violet Vaccines Containing Disodium Phosphate

Further experiments carried out in the Washington laboratories served to demonstrate that the germicidal action of crystal violet in defibrinated blood varies with the pH value of the blood. It was found that a slight increase in pH value was sufficient notably to enhance the germicidal action of crystal violet and that by rendering the blood slightly more alkaline through the addition of a small proportion of disodium phosphate, the desired degree of germicidal action could be obtained. Eleven lots of vaccine have been prepared by this method and five of these lots have been tested to date. Twenty-two pigs were used in these tests, ten receiving a 5-cc dose and twelve a 10-cc dose. All remained well following the vaccine treatment and all remained well when exposed to hog cholera at three weeks by virus injection. Therefore, in a limited number of tests (five lots of vaccine tested on 22 pigs), these vaccines have afforded 100 per cent protection and they are much superior in physical properties to vaccines containing phenol and orthocresol. More work will have to be done with these vaccines, however, before they can be evaluated properly.

Farm Experiments with Crystal-Violet Vaccines

One farm experiment has been completed and six others are under way on farms adjacent to the station. In these experiments, 360 pigs have been treated; these include suckling pigs weighing from 10 to 40 pounds and shotes weighing from 50 to 90 pounds. Seventeen of the suckling pigs were farrowed by susceptible sows; all the other pigs were from immune sows.

Regular vaccine, containing 0.1 per cent phenol and previously tested, was used on these herds. In the six experiments which are now in progress, a pooled vaccine, consisting of eight lots of previously tested vaccine, prepared between October 30, 1935, and January 14, 1936, was used. The pooled mixture was prepared on June 2, 1936, and the first of the six herds which received this vaccine was treated on June 12, 1936, and the last on August 7, 1936. The vaccine was administered by subcutaneous injection in 5-cc and 10-cc doses.

The first farm experiment to be completed will be described in detail. The farmer was advised that the vaccine treatment was still in the experimental stage, but he was quite willing to coöperate, as he had done before in other experimental work. Two pigs were purchased, brought to the station, injected with virus and found to be susceptible. This was done in order to establish the susceptibility of the herd. On November 5, 1935, the entire herd, consisting of 40 shotes weighing from 50 to 90 pounds and seven old sows, was treated with crystal-violet vaccine, each animal receiving 10 cc of the vaccine. The old sows had never been immunized against hog cholera and were given the same treatment as the shotes. All the pigs remained well after the vaccine treatment. Four months after treatment, five of the animals were purchased and brought to the station and exposed by virus injection. At four and one-half months after treatment, five more were obtained and exposed in like manner. At five months after treatment, another group of five was exposed and at five and one-half months still another group of five was exposed in the same manner. These animals weighed from 200 to 250 pounds at the time of exposure. Caution was exercised in exposing many at a time, in order to guard against possible financial loss, but as it turned out, fears on this score were groundless, for 17 of the 20 animals which were exposed remained normal after exposure, while three showed only a slight reaction and recovered promptly. The old sows in this herd were not exposed.

INTERVAL REQUIRED FOR THE ESTABLISHMENT OF IMMUNITY

Sixteen susceptible pigs of uniform size and weight, all obtained from one farm, were treated with the same lot of regular vaccine, each pig receiving a dose of 10 cc, administered subcutaneously. These pigs were exposed by virus injection at various intervals with the results shown in table I.

In this experiment, the results of which were very clear-cut, the immunity induced by crystal-violet vaccine had not been

established at one week, but was apparently complete at two weeks. The experiment also demonstrated that the immunity induced by the vaccine remained firm at the end of three months. In addition to the two pigs just mentioned, which were exposed at two weeks, four more vaccine-treated pigs were exposed at two weeks, making a total of six pigs exposed at this interval. One of these pigs had a severe reaction following exposure and recovered in fair condition, while the five others remained well. The fact that one pig, exposed at two weeks, suffered a severe reaction and recovered in only fair condition would seem to indicate that an interval of more than two weeks may be sometimes necessary for the establishment of immunity and led to

TABLE I—*Results of immunity tests at intervals after vaccination.*

PIGS	TREATMENT	RESULTS
2	Virus and vaccine at same time	Contracted hog cholera and died, or killed in worthless condition
2	Virus 1 week after vaccine	Contracted hog cholera and died, or killed in worthless condition
2	Virus 2 weeks after vaccine	Remained well
2	Virus 3 weeks after vaccine	Remained well
4	Virus 2 months after vaccine	Remained well
4	Virus 3 months after vaccine	Remained well

the decision that it would not be safe, in the course of our experimental work, to expose vaccine-treated pigs under three weeks. More experimental work is needed on this point, however.

COMPARISON OF SUBCUTANEOUS AND INTRAPERITONEAL ADMINISTRATION OF VACCINE

Thirty-two susceptible pigs were divided into two groups. One group was given subcutaneous injections of vaccine and the other group received intraperitoneal injections. The same vaccine was used on both groups, half of the pigs in each group receiving a 5-cc dose and the other half a 10-cc dose. Three weeks after receiving vaccine treatment, these pigs were exposed by virus injection with the results shown in table II.

The results following intraperitoneal administration were so poor in comparison with subcutaneous administration that further use of the intraperitoneal method was abandoned.

DURATION OF IMMUNITY FOLLOWING VACCINE TREATMENT

In addition to the 20 vaccine-treated shotes which were exposed to hog cholera in the farm experiment after intervals

ranging from four to 5½ months and found to be immune, 28 vaccinated station pigs were exposed by virus injection as follows: four at two months, four at three months, four at four months and 16 at six months. All remained well following exposure. While these data are not extensive, they indicate that the immunity following vaccine treatment persists for at least six months.

TABLE II—*Results of tests of immunity following two methods of vaccination.*

RESULTS	VACCINE INJECTED	
	SUBCUTANEOUSLY	INTRAPERITONEALLY
Remained well	15	8
Slight reaction	1	1
Severe reaction	0	6
Deaths	0	1

RETENTION OF POTENCY UNDER COLD STORAGE

Different lots of vaccine, which had been held under cold-storage conditions for varying lengths of time, were tested for potency. Fifteen lots were tested. Five were prepared with crystal violet alone and ten were regular vaccines, prepared with crystal violet and phenol (0.1 per cent). Two pigs were used in testing each lot. The storage periods for the vaccines ranged from 4½ to 14½ months. The vaccine dose was 10 cc and exposure by virus injection was made at three weeks after the vaccine treatment. All the pigs in these tests withstood the virus injection in good condition, 23 showing no reaction whatever and seven a slight reaction with quick return to normal. These tests indicate that crystal-violet vaccines may retain protective value for more than a year under cold storage, but there is need for further tests of vaccines stored for longer periods and used in smaller doses. The keeping qualities of crystal-violet vaccine containing disodium phosphate have not been determined.

EFFECT OF ANTI-HOG CHOLERA SERUM ON VACCINE IMMUNIZATION

In view of the considerable interval necessary for the development of immunity following vaccine treatment, it is obvious that certain contingencies could arise in connection with the field use of the vaccine when it might be desirable to supplement or follow up the vaccine treatment with anti-hog cholera serum. Experiments were planned accordingly, with a view to

determining whether the administration of serum would interfere with the antigenic action of the vaccine. One experiment has been completed in which 18 susceptible pigs were given vaccine and serum treatment (single or simultaneous) and exposed by virus injection at the end of three months. The object in holding these pigs for three months was to afford time for the passive immunity to lapse. The results of the exposure or immunity tests are shown in table III.

TABLE III—*Effect of anti-hog cholera serum on vaccine immunization.*

PIGS	TREATMENT	RESULTS*
2	Vaccine alone	Remained well
2	Serum alone	Developed hog cholera and were killed in worthless condition
4	Vaccine and serum at same time	Developed hog cholera and died or were killed in worthless condition
4	Vaccine followed in 1 week by serum	One died of hog cholera; others showed slight reaction but recovered in good condition
4	Vaccine followed in 1 week by serum and virus	Remained well
2	Untreated controls	Developed hog cholera and were killed for virus on eighth day

*Following virus injection at end of three months.

This experiment indicates that when vaccinated pigs are given the usual protective dose of anti-hog cholera serum, either simultaneously with the vaccine or within seven days after vaccination, the antigenic property of the vaccine is inhibited by the serum. The experiment is being repeated, as it throws light upon an important point in connection with vaccine administration.

DISCUSSION AND SUMMARY

Sixty-three lots of vaccine were prepared with crystal violet by several methods and tested on a total of 271 pigs.

In order to test their immunity, the vaccine-treated pigs were exposed to hog cholera by virus injection, which is undoubtedly a much more severe exposure than would ever occur under field conditions. Of the 271 vaccine-treated pigs, which were thus subjected to a severe exposure, 266, or rather better than 98 per cent, proved to be adequately protected against hog cholera. The injection of virus caused a slight reaction in about 9 per cent of the treated animals. This reaction, which occurred, as a rule, between the fourth and eighth days and lasted only

two or three days, was followed by a quick return to normal. The animals were not really sick, but were only a little slow in taking their feed. The reaction was usually so slight that it would have escaped notice in most cases, but for the fact that each animal was carefully watched at feeding time. In a farm herd, these slight reactions would in all likelihood have passed unnoticed and it has seemed proper to regard these pigs as having been adequately protected against hog cholera by the vaccine injections they received prior to exposure.

One farm experiment has been completed in which 40 shotes weighing from 50 to 90 pounds were given 10 cc each of vaccine. Half of this herd was exposed later by virus injection, after the animals had attained a weight of from 200 to 250 pounds. The results in this experiment were perfect, that is to say, all of the exposed animals were found to be adequately protected against hog cholera. Six more farm experiments are now under way, making a total of 360 pigs treated on farms. When completed, these experiments should furnish valuable data on the use of crystal-violet vaccines under field conditions.

It would seem to require between two and three weeks for the immunity to become established following vaccine treatment. This delay in the establishment of immunity will naturally limit to some extent the value of the vaccine in practice.

The duration of the immunity following vaccine treatment has not been established, but was found to be at least six months.

The keeping qualities of crystal-violet vaccines were tested after various periods in cold storage and no apparent deterioration was noted within 14 months.

The antigenic value of the vaccines was tested on pigs weighing from 40 to 90 pounds in 5-cc and 10-cc doses, and the smaller dose appeared adequate for complete protection.

The subcutaneous and intraperitoneal methods of administering vaccines were compared, and the subcutaneous method found to be notably superior.

Limited experimentation would seem to indicate that the usual protective dose of anti-hog cholera serum, administered to vaccine-treated pigs either simultaneously with the vaccine or within seven days after vaccination, inhibits the antigenic action of the vaccine and prevents the establishment of an active, permanent immunity.

While the results obtained thus far with crystal-violet vaccines have been excellent, these vaccines must be regarded as still in the experimental stage and much additional experimental work

remains to be done before their value is fully determined. It is hoped that the biological houses having suitable laboratories and qualified technicians, which have undertaken work on crystal-violet vaccines, will be able to supply much valuable data in addition to those reported in this paper.

A public patent covering the product has been applied for.

Discussion

Dr. W. H. Boynton: After hearing this paper, I should like to add a word of praise for Dr. McBryde, for the late Dr. Dorset, and for their associates on the progress they have made in the production of an active immunizing agent against hog cholera through the use of hog cholera virus modified by the crystal-violet method.

I feel, and most of us who have worked with hog cholera should agree, that the use of a product which eliminates unmodified virus from the field will be of great assistance in controlling the spread of hog cholera infection, as well as minimizing losses from intercurrent infections which occur so frequently following immunization by the serum-virus method.

In 1924, I began work on the development of a hog cholera tissue vaccine which would be a non-infectious immunizing agent. The method on which I have reported favorable results at two previous meetings of this Association involves the use of eucalyptol as the attenuating agent.

With the coöperation of the Cutter Laboratories, of Berkeley, California, I have continued work with my vaccine during the past year, and am conducting extensive field immunization trials. At the present time, approximately 1,400 weanling pigs have been treated, and results to date appear to be very satisfactory.

The owner of most of the pigs used in the field experiment has, for a number of years, suffered severe losses from enteritis and pneumonia, following immunization with serum and virus. Since we have been using the tissue vaccine for immunization purposes, the losses have been markedly reduced. From time to time, small groups of these pigs, immunized in the field, are brought to the laboratory and are tested for immunity by injecting them with hog cholera virus. Practically all of the pigs immunized in this way proved to be solidly immune to hog cholera.

I believe that real progress is being made in the development of a safe hog cholera vaccine, and hope it will not be long before such an agent will be available for general use.

Two little fellows were gazing at a zebra at the zoo.

"What a funny animal!" said one. "What is it?"

"I don't know," replied the other. "It's a sports-model donkey, I think." —*Our Dumb Animals*

"Did you hear about the cow that ate Kentucky blue grass?"

"No."

"Mood indigo." —*Phoenix*

REPORT OF AN OUTBREAK OF EQUINE INFECTIOUS ANEMIA, WITH OBSERVATIONS ON BLOOD CHANGES*

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Equine infectious anemia has existed in this country for at least 50 years, possibly much longer. It has, however, been known by various names: malarial fever, bottom-disease, swamp fever, and infectious anemia. Although the disease has been investigated extensively in some of the western states, the only published report of infectious anemia in New York State was made by Udall and Fitch,[1] in 1915. The outbreak occurred on the Saint Regis Indian Reservation, in Saint Lawrence and Franklin counties. Stein[2] recently stated that during the five years preceding 1929, infectious anemia occurred in 17 states, that the disease was reported as decreasing and of little or no economic importance in 14, while in three (Mississippi, Arkansas and Idaho) it was increasing and was an economic problem.

This report is presented to emphasize that infectious anemia does exist in the East, that it probably is not always recognized, that it presents a serious problem to institutions and individuals keeping a large number of horses, and that it can be controlled only by veterinarians being constantly on the alert for the appearance of symptoms and by employing adequate quarantine measures.

During the latter part of 1933 and the first quarter of 1934, ten definite cases of spontaneous infection and four following experimental inoculation were under observation at the laboratory farm. All suspected or recognized cases were placed in a screened infectious isolation unit, and no contact was permitted between the animals and attendants in this unit and those in the other buildings. Drainage was treated in underground chlorinating tanks before it entered the main sewer-line, and stable waste was burned. No known cases have appeared since.

The infection was first suspected in two horses undergoing experimental immunization against individual strains of streptococci. One horse had recurrent attacks of fever accompanied by a moderate decrease in the number of erythrocytes and an increased icterus index; the other, a greater reduction of erythro-

*Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

cytes and an equal degree of jaundice, but little or no elevation of temperature. The attacks had at first been attributed to immunization. A diagnosis of infectious anemia was made later in the first case. Both horses were destroyed.

In October, a horse which had not yet been assigned to immunization, developed a temperature of 106° F. ten days after it was brought in from pasture, and had symptoms and blood changes definitely suggestive of infectious anemia. Eight additional cases occurred within seven months. To confirm the diagnosis, five horses were inoculated subcutaneously with blood and intravenously with defibrinated blood or serum from definite or suspicious cases. Four developed the disease.

As it was important to eliminate the infection as rapidly as possible, 18 horses, including four not definitely diagnosed, were destroyed, the last on June 18, 1934. Hence, the natural outcome could not be observed. The chronic form of the disease predominated, although the frequency and severity of the attacks in a few cases more nearly corresponded to the subacute type.

Clinical Symptoms

Febrile attacks which occurred at more or less regular intervals and lasted for from two to nine days were characteristic of the infection. The average interval between attacks was 18.2 days, the shortest three, and the longest 40. The average length of 30 attacks in nine cases of spontaneous infection was 4.2 days, and of nine attacks in four inoculated horses it was 4.8 days. Morning temperatures were taken of all horses. Temperatures of the horses in quarantine were taken at least twice each day and usually at two-hour intervals during the fever periods.

In general, the beginning of an attack was manifested by a sudden onset of fever, which usually reached a maximum of from 104 to 107° F. during the first or second day. Partial loss of appetite, depression, languor and weakness occurred at that time. A pronounced unsteadiness of gait, especially in the hind quarters, and varying degrees of swaying and staggering frequently gave the animal the appearance of being about to fall. The weakness in the hind legs was further indicated by a marked dropping of the pasterns. There was a slight serous nasal discharge for two or three days. The mucous membranes of the eyes became pale and had a washed-out, yellow tinge which was most pronounced during the second or third day of fever. There was a moderate diarrhea in some cases. Usually, a pronounced loss of weight was observed during the attacks, which was later fully or partly regained.

Only one or two horses exhibited all of the symptoms; each of the others developed some of them in varying degree. The clinical manifestations at the time of the first febrile attack were frequently so mild that, in the absence of temperature readings and laboratory examinations, the illness might have been overlooked.

LABORATORY EXAMINATIONS

Icterus-index readings by Maue's modification of Meulengracht's method[3] were high, as would be expected following the reduction in number and the apparent destruction of the red blood-cells.

Comparative sedimentation tests were made by collecting 40 cc of blood from the jugular vein in graduated cylinders of uniform diameter, which contained 1 cc of a 20 per cent solution of potassium oxalate. Three readings were made: (1) the time required for the cells to settle to the 20-cc level (50 per cent separation), (2) the amount of sedimented cells at the end of 30 minutes (subtracted from 40 to give the amount of plasma or the distance of the fall), and (3) the amount of sediment at the end of 24 hours. The percentage of the total fall in 30 minutes was computed from the second and third readings. While the results of this procedure may be influenced by various factors, the large volume of blood and the simplicity of the technic tend to lessen certain errors encountered when working with small quantities. The results were consistent and sufficiently accurate for comparative purposes. They indicated that a decided acceleration in sedimentation took place during the attack and that, in a lesser degree, this increased rate in some cases continued for a time after the reëstablishment of the normal red-cell count.

In the majority of the cases, the speed of sedimentation was greatest when or soon after the fever reached its highest point; in some, after it had subsided. The minimum time for 50 per cent separation was usually less than ten minutes, in one case, five, as compared with the 30 to 60 or more minutes usually required for blood from healthy horses. Eighty-five to 95 per cent of the total fall was obtained in 30 minutes. The red blood-cell counts were considered in the interpretation of the rate of sedimentation, although actual corrections were not made on the basis of cell count or cell volume. While not specific, the acceleration of sedimentation forms part of the composite picture characteristic of the disease.

Routine blood-cell counts contributed largely to the early recognition of the infection. In some cases, daily blood examinations were made during and immediately after the febrile attacks and were followed by occasional later examinations. Frequent examinations during the period between the onset of fever, and from six to ten days after, furnished the most valuable information. This information afforded data not only on the degree of change in the blood, but also on the time of its occurrence. The latter offers an explanation of the marked variations noted in published reports of limited numbers of blood counts made at different points during the febrile period.

On the fourteen cases, 263 red blood-cell counts were made. Of those made during 28 attacks in the ten horses with spontaneous infection, minimum counts of less than 5,000,000 were obtained in twelve; in three, there were 7,000,000 or more red blood-cells. The greatest reduction occurred at the time of maximum temperature, or within from twelve to 24 hours of it. Five to seven days later, the number of red cells in some cases reached the pre-attack level, although in others a moderate degree of anemia was present between attacks. The anemia in many instances was no greater than that found in horses with other infectious processes or in those undergoing certain forms of immunization.

Our results indicated that there was a moderate reduction in the number of leukocytes, which became more apparent at the end of the febrile attack, and a distinct shift in the polymorphonuclear lymphocyte ratio. A transitory increase in polymorphonuclear cells, accompanied by a decrease in lymphocytes, indicated the beginning of the change. In general, the polymorphonuclear cells started to decrease at the time of maximum temperature and continued to drop rapidly until a day or two after the temperature returned to normal. At that time, in some cases, they were one-half or even one-fourth of the number present at the beginning of the fever. The loss of neutrophils contributed mainly to the lowered white-cell counts. The number of lymphocytes, which was low at the time of the highest temperature, or shortly thereafter, gradually increased until it reached the maximum, from two to four days after the temperature became normal. A relative lymphocytosis was a common finding.

Schermer, Eigendorf and Traupe[4] have reported that an increase in monocytes (from 4 to 9.6 per cent) is not uncommon in infectious anemia. The same was true during the present out-

break. Monocytes (large mononuclear and transitional forms) were, in general, at their highest within from one to three days after the return of normal temperature, and remained high for from two to five days. The average maximum monocyte count in 21 attacks was 842, or 9.2 per cent of the corresponding average white-cell count of 9,145.

In 69 cell counts made of the blood of 51 apparently healthy horses received at the laboratory, the average percentage of monocytes was 1.3; the highest in any one horse was 5.3. The observed increase in monocytes is not, in our experience, a common finding in horses undergoing immunization or in those suffering from other conditions. It was, however, after febrile attacks due to infectious anemia and it appeared, therefore, to have considerable diagnostic significance.

A temporary reduction in eosinophils, not uncommon at the time of high temperatures, was regularly observed in these cases. Frequently, no eosinophils could be found in counting 300 white cells.

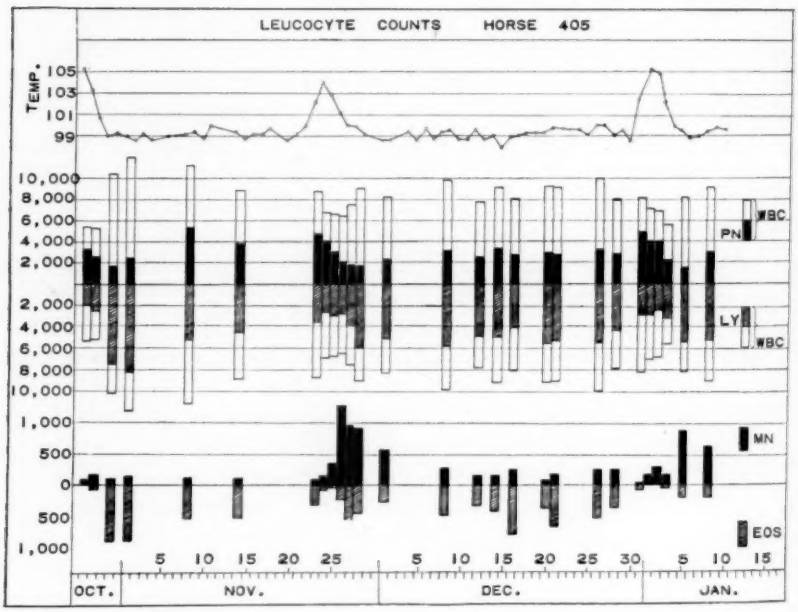

FIG. 1. Chart of horse 405. Leukocyte counts during and between fever periods: variations in total white blood-cell count, shift in polymorphonuclear lymphocyte ratio during the following fever, decrease in eosinophiles during fever, and increase in monocytes near the end of or following febrile attacks.

SUMMARY

This paper records the clinical observations and laboratory findings during a small outbreak of equine infectious anemia which occurred in New York State. Attention is called to the fact that this disease is not always recognized and that it presents a serious problem to owners of large groups of horses.

The changes in the blood and the time of their occurrence in relation to the elevation of temperature were carefully studied. A reduction was noted in the number of red blood-cells, and there was an increased icterus index which was most marked at the time of, or immediately following, the highest temperature. An acceleration in the rate of sedimentation of the blood-cells occurred and was greatest at the time of or following the maximum temperature. Little or no increase was noted in the number of white blood-cells at the onset of fever but during the febrile period the cells decreased frequently to a point below the normal level. This was due mainly to the reduced number of polymorphonuclear cells and was most noticeable near the end of the temperature period. Following the fever, the number of lym-

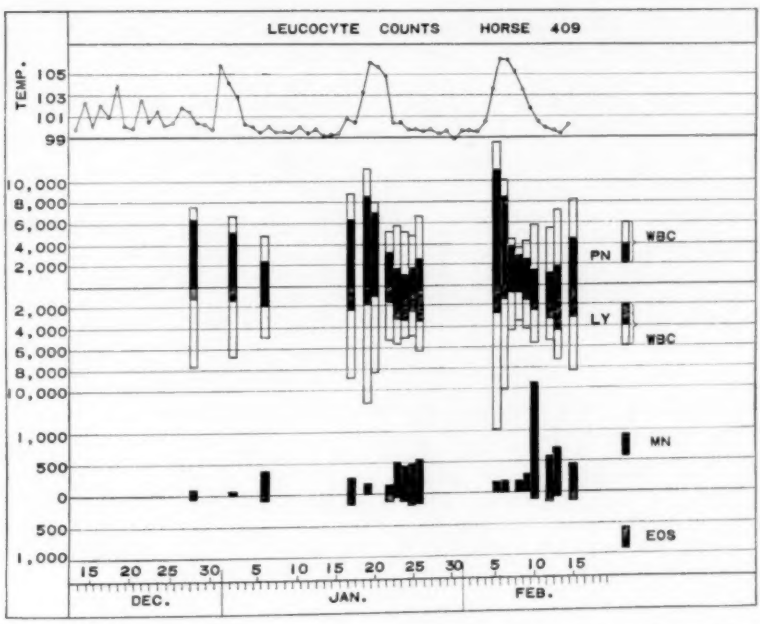

FIG. 2. Chart of horse 409. Leukocyte counts during and between fever periods: variations in total white blood-cell count, shift in polymorphonuclear lymphocyte ratio during the following fever, decrease in eosinophiles during fever, and increase in monocytes near the end of or following febrile attacks.

phocytes increased and frequently exceeded that of the polymorphonuclear cells. The temporary increase of large mononuclear and transitional forms after the period of fever was a characteristic finding which has not so far been noted in blood examinations of other horses under our observation.

REFERENCES

[1]Udall, D. H., and Fitch, C. P.: Preliminary report on the recognition of swamp fever or infectious anemia in New York State. Corn. Vet., v (1915), pp. 69-80.

[2]Stein, C. D.: Infectious anemia or swamp fever in horses. A review of the Bureau of Animal Industry's investigations. Jour. A. V. M. A., lxxxvii (1935), n. s. 40 (3), pp. 312-324.

[3]Maue, H. P.: Icterus index of blood serum. Surg. Gynec. & Obst., xxxiv (1922), pp. 752-754.

[4]Schermer, S., Eigendorf, R., and Traupe, H.: Hämatologische Untersuchungen bei der infektiösen Anämie und ihre diagnostische Bedeutung. Arch. f. Wiss. prakt. Tier., lvii (1928), pp. 445-471.

Horses and Mules on Farms Continue to Decrease

According to a forecast recently made by the U. S. Bureau of Agricultural Economics, in its Annual Outlook Report, a further decline in the number of horses and mules on farms may be expected during 1937 and for several years to come. The low point in the downward trend probably will not be reached for four or five years.

The low point in the number of animals of work age will occur a few years after the low point in numbers is reached. During this period, prices of horses and mules are expected to increase somewhat above present prices. Incident to the decrease in the number of work animals, an increase in the use of tractors is to be expected. The report continues:

> The extent to which tractors displace work stock in the next few years will not only affect prices for horses but will influence the number of animals needed for power on farms. With the non-farm outlet for work stock practically gone, and with large acreages of new lands for agricultural purposes no longer available, the need for work stock in the future will be much more limited than was the need for many years prior to 1918, when horse and mule numbers were increasing rapidly.
>
> Perhaps the maximum number needed for farm use is little different from the number now on farms. However, the need of replacing many old horses with younger animals is a factor tending to increase colt production. Demand has been principally for young animals, and especially for young mares suitable for both work and breeding.

The vow that binds too strictly snaps itself.—ALFRED TENNYSON.

THE UTILIZATION OF SHEEP IN THE RABIES VACCINE PROTECTION TESTS*

By J. E. SCHNEIDER, *Glenolden, Pa.*
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In 1934, Reichel and Schneider[1] reported the intralingual route for the administration of rabies virus. By the intralingual injection it was demonstrated that the efficacy of phenol-killed rabies vaccine could be measured with some degree of regularity.

Employing the subcutaneous, intraocular or intracranial route or injections into the lumbar plexus gave such variable results that it was difficult to draw definite conclusions. However, as previously reported[1] and substantiated by subsequent experimentation, the intralingual route for rabies virus administration has served as a suitable means for reaching reliable conclusions. By this method a particular strain of virus would produce results which were not only capable of repetition but a method was available for measuring the protection afforded in the use of rabies vaccine.

Our interest in these experiments has continued with an attempt to establish some usable method for estimating the value of phenol-killed rabies vaccine. As previously reported, protection tests on rabbits and dogs, followed by the administration of virus intralingually, demonstrated the protective value of rabies vaccine. Clinically the use of rabies vaccine has, by inference, been termed successful, but demonstrable value in larger animals has been lacking.

The experiments reported herein are the results of our experiences using rabbits and sheep as the test animals in the prophylactic administration of rabies vaccine and, following a period of immunization, the injection intralingually of the infective dose of virus. Two types of rabies vaccine were used in these protection tests. Vaccine A consisted of a 25 per cent brain and cord tissue, phenol-killed, prepared from rabbits, and vaccine B consisted of a 20 per cent brain and cord tissue, phenol-killed, prepared from horses.

Vaccine A: Twenty rabbits were injected with 0.1 cc of a 5 per cent brain tissue emulsion of fixed rabies virus intracranially, and when the animals became moribund with rabies, the brains and cords were removed and ground in a mortar and

*Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

pestle by hand under a sterile hood. The material was diluted to 50 per cent brain and cord tissue emulsion. Phenol was added to a concentration of 1 per cent, and held for 21 days in cold storage at 5° C., then placed in the incubator for three days at 37.5° C. for the purpose of killing the rabies virus. The vaccine was then diluted to a 25 per cent brain and cord tissue emulsion containing 0.5 per cent phenol.

Vaccine B: Five horses were injected with 0.1 cc of a 5 per cent brain tissue emulsion of fixed rabies virus intracranially and when the animals became moribund with rabies the brains and cords were removed, and ground in a ball-mill for 15 hours. The material was diluted to 50 per cent brain and cord tissue emulsion. Phenol was added to a concentration of 1 per cent, and held at cold storage temperature and shaken daily for 21 days, then placed in the incubator at 37.5° C. for three days, for the purpose of killing the rabies virus. The vaccine was then diluted to 20 per cent brain and cord tissue emulsion containing 0.5 per cent phenol.

The immunizing values of the two vaccines, A and B, prepared respectively from the rabbit and from the horse, were then determined as shown in tables I and II.

TABLE I—*Rabies vaccine protection test on rabbits.*

GROUP	RABBITS	RABIES VACCINE (PHENOL-KILLED)		INTERVAL BEFORE INFECTIVE DOSE* (DAYS)	RESULTS†
		KIND	AMOUNT		
1	6	A‡	1 dose (5 cc)	21	Living 100%
2	6		14 doses (0.5 cc each)	8	Living 100%
3	6	B§	1 dose (5 cc)	21	Living 83%
4	6		14 doses (0.5 cc each)	8	Living 100%
5	3		Intralingual controls		Dead 100%

*0.5 per cent brain tissue emulsion rabies virus 0.1 cc injected intralingually.

†Deaths preceded by definite symptoms of rabies.

‡25 per cent brain and cord tissue emulsion rabies vaccine (rabbit) No. 4278 injected subcutaneously.

§20 per cent brain and cord tissue emulsion rabies vaccine (horse) No. 4280 injected subcutaneously.

Table I shows that six rabbits were injected subcutaneously with a single 5-cc dose, and six with a 0.5-cc dose, daily for 14 days, of rabies vaccine prepared from rabbits (A). Twenty-one days after the injection of vaccine to the group of rabbits receiving the single 5-cc dose, and 8 days after the last dose in the group receiving a 0.5-cc dose daily for 14 days, an infective dose of fixed rabies virus was administered intralingually. All the test rabbits in both series survived, while 100 per cent of the control animals died.

Six rabbits were injected subcutaneously with a single 5-cc dose and six with a 0.5-cc dose daily for 14 days, with rabies vaccine prepared from horses (B). After an interval of 21 days, an infective dose of fixed rabies virus was given intralingually to the group of rabbits receiving the single 5-cc dose. Of these, 83 per cent survived. When the group of rabbits receiving a 0.5-cc dose daily for 14 days were given the infective dose of fixed rabies virus intralingually after an interval of 8 days, 100 per cent survived, while 100 per cent of the controls died.

TABLE II—*Rabies vaccine protection test on sheep.*

GROUP	SHEEP	RABIES VACCINE B (PHENOL-KILLED)*	INTERVAL BEFORE INFECTIVE DOSE† (DAYS)	RESULTS‡
1	6	1 dose (5 cc)	21	Living 83%
2	6	2 doses (5 cc each)	20	Living 100%
3	6	6 doses (5 cc each)	15	Living 83%
4	6	Intralingual controls		Dead 100%

*20 per cent brain and cord tissue emulsion rabies vaccine (horse) No. 4280 injected subcutaneously.
†0.5 per cent brain tissue emulsion rabies virus 0.5 cc injected intralingually.
‡Deaths preceded by definite symptoms of rabies.

Table II shows the results of rabies vaccine protection tests on sheep. The same rabies vaccine, 20 per cent brain and cord tissue emulsion from horses (B), used in table I, was used with the test on sheep.

Eighteen sheep were treated, six of them being given one 5-cc dose; six sheep two 5-cc doses of vaccine, one day apart; and six sheep six 5-cc doses at daily intervals. All sheep were given an

infective dose of fixed rabies virus intralingually 21 days after the first immunizing dose with the following results:

Of six sheep injected with a single 5-cc dose, with a 21-day interval, 83 per cent survived.

Of six sheep injected with a 5-cc dose daily for two days, with a 20-day interval, 100 per cent survived.

Of six sheep injected with a 5-cc dose daily for six days, with a 15-day interval, 83 per cent survived.

Of the six control sheep, 100 per cent died.

SUMMARY

The use of the intralingual method of administering the infective dose of rabies virus in our hands has given consistent results in the protection test of rabies vaccine on rabbits.

Our results on sheep show that the use of the intralingual method is suited for this type of protection test.

It was our experience in making these tests that sheep were more satisfactory than dogs for test purposes because they require less care and, more important, their health is less impaired during the confinement of the test period.

In view of our observations, sheep may prove to be the best medium for testing the potency of rabies vaccine.

The results shown in table I show that a single 5-cc dose of rabies vaccine B containing 20 per cent brain and cord tissue protected five out of six rabbits against an infective dose injected intralingually. This same infective dose administered by the same route, however, killed 100 per cent of the controls. It is shown also in table II that a single 5-cc dose protected five out of six sheep against an infective dose injected intralingually, which killed 100 per cent of the controls. Although the high degree of immunity produced was not sufficient to protect all the rabbits and sheep against the severe exposure of fixed rabies virus injected intralingually, we feel it to be ample against any natural mode of exposure.

In conducting a rabies protection test it must be remembered that rabies virus is neurotropic and the success of the test depends upon the method of injecting the infective dose. In addition, the rabies virus injected must be virulent and in adequate amounts to produce rabies in at least 66 per cent of the controls. The infectivity of rabies virus cannot be determined before it is used, because the virus has to be freshly prepared. Therefore, it is to be expected that the virus used as the infective dose will vary to such an extent that it may kill up to 100 per

cent of the controls. When this high rate of mortality occurs among the controls, the animals under test are likewise exposed.

Any experiment in which the infective dose is of sufficient virulence to kill 100 per cent of the controls (tables I and II) places severe exposure on the test animals.

It is our belief that the rabbits (table I) and sheep (table II) in these experiments which showed 83 per cent protection would have been 100 per cent protected against an infective dose which killed 66 per cent of the controls.

Rabies vaccine injected in a single 5-cc dose which protects 66 per cent of the test animals against an infective dose of rabies virus which kills 66 per cent of the controls is, in our opinion, a good vaccine, and should afford ample protection against natural exposure.

In table II (group 3) the administration of 5 cc of rabies vaccine B daily for 6 days protected five out of six sheep against an infective dose injected intralingually which killed 100 per cent of the controls.

The results confirm our contentions expressed in a previous article that with injections of multiple doses of rabies vaccine, the interval between the last dose of rabies vaccine and the injection of the infective dose of virus is an important factor in obtaining maximum protection. Had the experiments been repeated with the same interval, for example, 21 days following the last injection of vaccine, a higher degree of protection might have resulted. It should also be borne in mind that the quantity of brain and cord tissue vaccine injected is essential for maximum protection under the conditions of these experiments.

It is a well established biological fact that multiple injections of an antigen afford a higher degree of immunity than single injections. These experiments add further evidence to emphasize the value of repeated injections. One 5-cc injection in rabbits gave 83 per cent protection, whereas a 0.5-cc dose administered daily for 14 consecutive days afforded complete protection.

Conclusions

It has been shown that rabies vaccine A, containing 25 per cent rabbit brain and cord tissue, protected rabbits in a single 5-cc dose or in 14 doses of 0.5 cc each at daily intervals.

The tests also show that rabies vaccine B, containing 20 per cent horse brain and cord tissue, protected rabbits equally as well as the rabbit vaccine A in the group injected with 14 doses of 0.5 cc each at daily intervals. The group of rabbits injected

with a single 5-cc dose of vaccine A were 100 per cent protected, whereas with vaccine B one rabbit out of six died.

Rabies vaccine B, containing 20 per cent horse brain and cord tissue, showed a high degree of protection in each of the three groups of sheep (table II).

Results as shown should not be strictly interpreted to apply to field conditions for the reason that in conducting a test of this kind the animals are exposed in an unnatural way, and with an undoubtedly greater amount of rabies virus than would be met with under natural exposure.

These tests show the degree of protection produced depends on the amount of brain and cord tissue injected, whether it be in a single dose or in multiple doses, together with the length of time between the last dose of vaccine and the injection of the infective dose. In other words, maximum protection is an accumulation of the size of the dose, distributed over several days and allowing a sufficient interval before the infective dose.

Reference

[1]Reichel, J., and Schneider, J. E.: Rabies vaccine protection test. Jour. A. V. M. A., lxxxiv (1934), n. s. 37 (5), pp. 752-758.

Discussion

Dr. H. W. Schoening: Since the introduction of the single-injection rabies vaccines in this country for prophylactic use in controlling rabies in dogs, there has been considerable discussion as to the efficacy of this product. Experimental work on the subject has been undertaken by various investigators and reports of these works have been published. The results have been variable.

Many difficulties are encountered experimentally in testing the protective properties of rabies vaccines since the exposure virus and the method of exposure to which the vaccinated animals are subjected are factors of great importance in evaluating the results obtained. Since rabies vaccines are primarily intended for use on the dog, experimental work using dogs as test animals has received the most attention.

In testing the potency of rabies vaccines on dogs, difficulties are encountered in finding a suitable method of exposure. Natural exposure in which dogs are subjected to the bites of a rabid dog, at first sight, may seem to furnish an ideal method of testing immunity since this is the method by which the disease is transmitted naturally. However, the irregularity with which normal dogs contract rabies after being bitten by rabid dogs makes this method unreliable. Moreover, the exposure that each dog receives by this method cannot be regulated; one dog may receive more bites than another while the location of the wounds inflicted may be quite different.

The intracerebral and the intraocular injections of virus are effective means of producing the disease but are open to the criticism that they are too severe a test in that infection so transmitted is probably much greater than would occur under average natural conditions. Since the immunity produced by any biological product is relative, it is quite readily seen that immunity might be broken down by extreme exposure.

The subcutaneous injection of street virus is quite irregular in its action. Better results have been obtained with intramuscular injections, particularly in the region of the loin, but the results at times are quite irregular. The same difficulties in finding a suitable method of exposure for laboratory animals have made the potency testing of rabies vaccines on these animals a difficult procedure.

Reichel and Schneider, at the meeting of this Association in Chicago, in 1933, reporting on protection tests in rabbits with rabies vaccines, described the intralingual method of exposure, which consists of an injection into the muscles of the tongue of 0.1 cc of a 5 per cent suspension of either fixed or street virus. They reported that 66 to 100 per cent of the animals so treated developed the disease.

Since that time, the Pathological Division of the Bureau of Animal Industry has worked with this method of exposure in rabbits and has been able to verify fully the results of Reichel and Schneider as to its effectiveness. It has been our experience that infection occurred regularly in from 70 to 100 per cent of the rabbits injected intralingually with either fixed or street virus.

Using this method of exposure, we have tested the potency of a number of chloroform-killed rabies vaccines of our own manufacture and have found that the method lends itself to this purpose. Against the intralingual method of exposure, we have been able, in limited tests, to protect in some instances six rabbits which had received a 5-cc injection of vaccine whereas of an equal number of control animals, all died of rabies.

From the excellent results of the protection tests reported in Dr. Schneider's paper today, using sheep as test animals, further encouragement is given to the hope that a satisfactory method of testing rabies vaccines is now available and it is to be hoped that extensive trials of the method, using dogs as test animals, will be made by agencies properly equipped.

Kansas State College on the Air

Members of the staff of the Division of Veterinary Medicine at Kansas State College are scheduled to go on the air in a series of radio talks from Station KSAC during the early months of 1937. The talks will be given at 5:00 p. m., C. S. T. Station KSAC operates on a frequency of 580 kilocycles. The following schedule has been announced:

February 5—"Timely Veterinary Suggestions," Dr. E. J. Frick.
February 12—"Progress in Veterinary Medicine," Dr. J. H. Burt.
February 19—"Preparing the Horse for Spring Work," Dr. E. R. Frank.
February 26—"Pasture Losses in Cattle," Dr. H. F. Lienhardt.
March 5—"Feeding the Family Pet Animal," Dr. E. P. Leonard.
March 15—"Bang's Abortion Disease in Cattle," Dr. C. H. Kitselman.
March 22—"The Value of Medicines," Dr. R. P. Link.
March 29—"Cattle Anaplasmosis in Kansas," Dr. Herman Farley.
April 5—"Champion Obstacle Racers," Dr. J. H. Whitlock.
April 12—"Too Much Growth," Dr. C. C. Morrill.
April 19—"Adequate Water for Farm Animals," Dr. E. E. Leasure.
May 3—"Comparing the Human and Animal Skulls," Dr. W. M. McLeod.
May 10—"The Veterinarian Guards Human Health," Dr. R. R. Dykstra.
May 17—"Hygiene in Pure Bred Herds," Dr. C. D. Ebertz.

ONE THOUSAND COWS TESTING SUSPICIOUS (1:50) TO THE BANG AGGLUTINATION TEST AND THEIR SUBSEQUENT TEST BEHAVIOR*

By CHAS. H. KITSELMAN, *Manhattan, Kan.*
Kansas Agricultural Experiment Station

Workers with Bang's abortion disease of cattle have for many months pondered the question as to the importance of the Bang suspect in herd testing. Some have preferred to ignore the animal unless it showed advanced pregnancy. Others insisted that the reaction of 1:50 was evidence of infection and consequently the animal should at least be segregated from non-reacting cattle.

For the past five years, we have followed the policy of allowing the suspect to remain in the herd subject to a retest in 30 days, if not pregnant, and in many instances have found that the animal was either distinctly positive or distinctly negative on the retest.

When the opportunity presented itself to study 1,000 breeding cattle tested in the same laboratory by the same technician over a definite period of time, the data from the group were tabulated for an exhaustive study. In studying the herds, particular attention was paid to the amount of infection found on the first test. In many cases the suspects were from unrelated groups. For ease in tabulating results, the state average of 15 per cent was taken as an arbitrary standard and the herds placed in groups. All reacting animals were promptly removed from the herds. The herds were grouped as follows:

Group I: Initial infection 15 per cent or higher, and reactors found in the herd nine months later.

Group II: Initial infection 15 per cent or higher, as in above group, but no reactors found in the herd nine months later.

Group III: Initial infection less than 15 per cent but herd still contained reactors at end of nine months.

Group IV: Initial infection similar to group III, but no reactors found in herd at end of nine months.

In table IV, groups I and II are combined with a total number of 649 animals and an average initial herd infection of 17.45 per cent. Groups III and IV are combined with a total of 351 animals and an initial herd infection of 11.2 per cent.

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*Contribution No. 68 from the Department of Veterinary Medicine, Kansas State College. Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

TABLE I—*Distribution of suspects in the arbitrary groups.*

GROUP	SUSPECTS	INITIAL HERD INFECTION (%)	HERD STILL INFECTED IN NINE MONTHS
I	416	17.9	Yes
II	233	17.0	No
III	220	12.3	Yes
IV	131	10.1	No
Totals	1,000		

TABLE II—*Results of retest at the end of five months of the 1,000 suspects listed in table I.*

GROUP	REMAINED SUSPECTS		BECAME NEGATIVE		BECAME POSITIVE	
	No.	%	No.	%	No.	%
I	48	11.5	197	47.4	171	41.1
II	16	6.8	217	93.2	—	—
III	93	42.3	67	30.4	60	27.3
IV	11	8.4	120	91.6	—	—
Totals	168		601		231	

TABLE III—*Results of a second retest, at the end of nine months, of the 168 suspects listed in table II.*

GROUP	REMAINED SUSPECTS		BECAME NEGATIVE		BECAME POSITIVE	
	No.	%	No.	%	No.	%
I	1	0.59	20	11.91	27	16.08
II	—	—	16	9.51	—	—
III	—	—	86	51.19	7	4.17
IV	—	—	11	6.55	—	—
Totals	1		133		34	

TABLE IV—*Distribution of suspects in combined groups.*

GROUPS	AVERAGE INITIAL HERD INFECTION (%)	SUSPECTS (%)	FIVE-MONTH TEST			FIVE-MONTH TEST		
			POSITIVE (%)	SUSPECTS (%)	NEGATIVE (%)	POSITIVE (%)	SUSPECTS (%)	NEGATIVE (%)
I and II	17.45	64.9	26.4	9.8	63.8	42.2	1.54	56.26
III and IV	11.2	35.1	17.6	29.3	53.1	6.73	—	93.27

Conclusion

1. At the expiration of five months, 60.1 per cent of the cattle which had reacted in a dilution of 1:50 had become definitely negative to the test and 23.1 per cent had become definitely positive in a dilution of 1:100 or higher, whereas 16.8 per cent remained unchanged in the suspicious classification.

2. Approximately 41.6 per cent of the suspicious animals on the initial test were in the group of herds having 17.9 per cent infection, 23.3 per cent were in the group having 17.0 per cent infection, 22.0 per cent were in the group of herds having 12.3 per cent infection and 13.1 per cent were in the group showing 10.1 per cent initial infection.

3. It seems, therefore, that practically all animals found to be reactors in a dilution of 1:50 on the initial test will definitely become either negative or positive within a period of nine months, and many within a period of five months.

4. These data may be of use in advising herd-owners regarding the disposition of certain animals which react in the 1:50 dilution when the herd test history is available.

Discussion

Dr. R. R. Birch: I have just one point with respect to Dr. Kitselman's paper. He did keep the records on one of the suspicious reactors and the number of suspicious reactors which were subsequently positive and subsequently negative. I wonder if he kept records of similarly exposed animals which were negative on the initial test?

Dr. E. J. Frick: Yes. He has all those records, because most of these herds are pure-bred, private herds which he has been taking care of for a long time.

World's Poultry Congress Committee Appointed

Under date of November 17, 1936, the appointment of a committee to represent the U. S. Department of Agriculture in plans for the Seventh World's Poultry Congress was announced, as follows:

Berley Winton, Bureau of Animal Industry, chairman; W. D. Termohlen, Agricultural Adjustment Administration, secretary; Robert R. Slocum, Bureau of Agricultural Economics; H. L. Shrader, Extension Service; T. L. Swenson, Bureau of Chemistry and Soils; Ernest G. Moore, Office of Director of Information; Ruth Van Deman, Bureau of Home Economics.

The committee will work with representatives of other departments of the government and with committees representing various interests identified with the poultry industry.

A STUDY OF TRANSMISSIBLE FOWL LEUKOSIS*

By CARL OLSON, *Rochester, Minn.*
The Mayo Foundation

Fowl leukosis has been the subject of considerable study during recent years. Various opinions have been advanced on the relationship between certain diseases designated as erythroleukosis, myeloid leukosis, lymphocytoma, myelocytoma, neurolymphomatosis gallinarum, endothelioma and sarcoma. A discussion of these relationships will be presented in a later report on a review of the literature.

The data to be presented in this report concern observations made on three strains of transmissible leukosis. Especial attention was given to the changes in the blood of the experiment animals during the course of the disease. Certain observations on the pathologic changes involved in the disease process also were made. It seemed advisable to establish certain factors of similarity between the strains of disease studied and those strains of disease reported by others. Some of these factors which were examined were the species specificity of the transmissible agent, its filtrability, and its vitality in glycerin solution.

METHODS

The experiment chickens were hatched and raised at the Institute of Experimental Medicine. Most of the chickens were Barred Plymouth Rocks. Adult members of the flock of Barred Plymouth Rocks maintained at the laboratory were not infrequently observed to develop spontaneously erythroblastic or granuloblastic leukosis. Apparently there was some inbred factor that accounted for this incidence of spontaneous leukosis. Among flocks of other breeds of chickens maintained at the laboratory, the incidence of spontaneous disease was not so high. Spontaneous fowl leukosis was never observed in a Barred Plymouth Rock of less than six months of age. Hence, by using younger chickens, the advantage of a known susceptible strain of chickens was obtained and the factor of spontaneous occurrence of disease in experiment animals was practically eliminated. Other breeds of chickens used were white Leghorns, and crossbred chickens hatched from eggs laid by White Leghorn hens mated to Barred Plymouth Rock roosters. The majority of the

*Abridgment of a portion of thesis submitted to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

chickens were two months of age or less at the time of inoculation.

These chickens were housed in cages. Usually chickens inoculated with similar material were kept in the same cage. The dietary regimen was essentially the same for all of them.

Transmission of the disease was in most instances effected by the use of whole blood obtained from the sick fowl. The blood was withdrawn from the wing vein into a syringe containing an anticoagulant (heparin, sodium citrate, or potassium oxalate). Emulsions of tissue were used in some instances. These were prepared by grinding the tissue with sterile, normal salt solution. The resultant emulsion then was filtered through gauze to remove the coarser particles. Intravenous inoculations were made in most instances into the wing vein. For very small chickens the jugular vein was used.

Routinely, a blood smear, and usually total leukocyte, thrombocyte, erythrocyte, and differential counts and a hemoglobin determination, were made for each chicken before it was inoculated. These data were used to determine the fact that the chickens were normal in these respects at the time of inoculation. Blood studies were made at intervals of six to ten days during the experiment. The observations included an erythrocyte count, hemoglobin reading, and a determination of the relative numbers and character of pathologic blood-cells seen in the blood-smear.

The erythrocyte diluting fluid used in making the counts was that reported by Wiseman,[1] utilizing the dye, phloxine. This was found to be very adaptable for the study of pathologic blood (Olson). The determinations of hemoglobin were made with the Sheard and Sanford photoelectric hemoglobinometer.[2] The blood-smears were for the most part stained with Wright's blood stain. In some instances the May-Grünwald-Giemsa technic was used.[3]

The grading system used for estimation of the character of the leukemic blood is discussed in the section dealing with changes in the blood.

All chickens were examined at necropsy and observations were made on the pathologic changes present. Tissues (routinely portions of liver, spleen, femoral marrow, and sometimes other tissues) were taken for later histologic examination. Usually these were fixed in neutral 10 per cent formalin solution and stained with hematoxylin and eosin. The tissues from certain animals for special study were fixed in Helly's fluid and stained either with hematoxylin and eosin or by the Dominici technic.

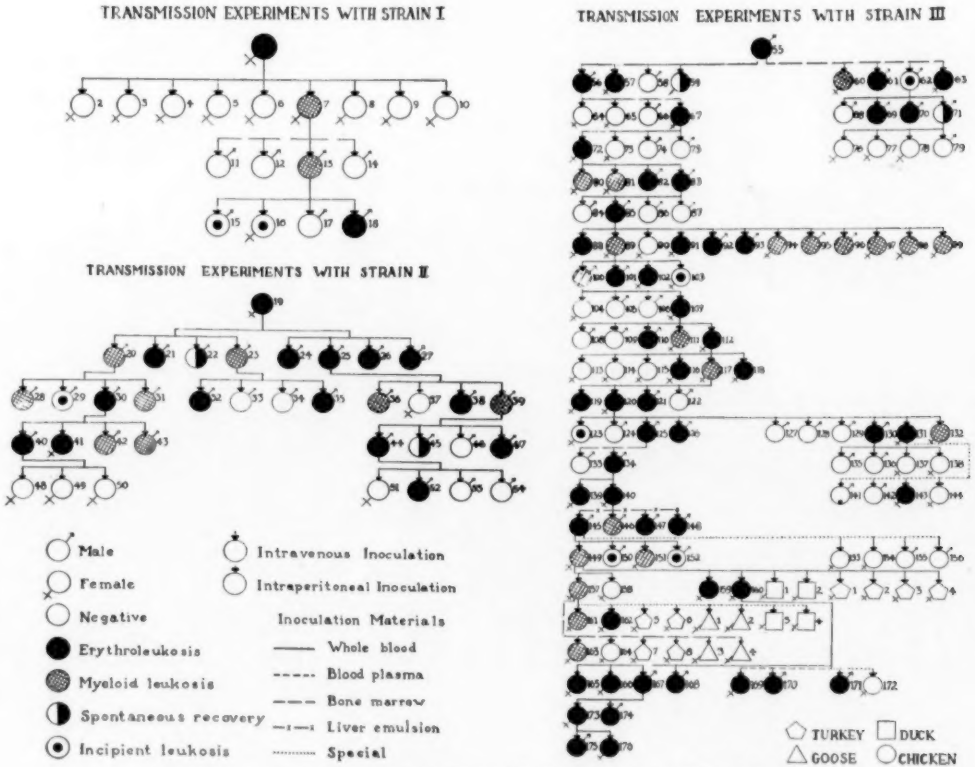

FIG. 1. Transmission experiments with three strains of fowl leukosis.

Description of Transmissible Strains

Origin: The three strains of transmissible leukosis were obtained from cases of spontaneous fowl leukosis which occurred among the chicken population at the Institute of Experimental Medicine.

A Barred Plymouth Rock hen (chicken 1, fig. 1), aged 20 months, was the source of strain I. This chicken was listless, and the comb, wattles and legs had an anemic appearance. Examination of the blood revealed the following data: erythrocytes, 360,000; thrombocytes, 13,250; mature leukocytes, 11,900, and immature blood-cells, 20,400 per cmm. The value for hemoglobin was 0.25 gm per 100 cc of blood. The differential count of the mature leukocytes revealed the following percentages: lymphocytes, 42.5; heterophils, 32.5; eosinophils, 5.0; basophils, 6.5, and monocytes, 13.5. After removal of whole blood for the inoculation experiments, the chicken was killed for necropsy. The gross and microscopic pathologic findings were those typical of erythroblastic leukosis. These pathologic changes are described in more detail in another section of this report.

Transmissible strain II likewise had its origin in a Barred Plymouth Rock chicken. This bird (chicken 19, fig. 1) was 17 months of age when first observed. It was one of the group selected for inoculation with the leukemic blood from chicken 1 which was the source of strain I. Chicken 19 manifested no outward signs of disease at the time of inoculation. Specimens of blood were taken and it was inoculated with 1 cc of whole blood from chicken 1. This was done before the specimens of blood were examined. Subsequent examination of these specimens revealed the fact that the chicken had already begun to show changes characteristic of leukosis before receiving the inoculation from chicken 1. It was therefore evident that this bird had the disease at the time of inoculation. The effect of inoculation of leukemic blood into this animal is not known. When the course of the disease was compared with that of experimental leukosis, no specific differences were evident. The data on this fowl seem to indicate that, when first observed, it was in the beginning stages of the disease. I believe that this is the first spontaneous case of leukosis to be observed from the time of the initial symptoms to the time of death. When first observed, the chicken weighed 1,613 gm; at the time of its death 87 days later, it weighed only 860 gm. This loss of weight was gradual. The blood changes observed during the course of the disease are given in table I.

TABLE I—*Changes in the blood in a case of spontaneous fowl leukosis (chicken 19).*

DAYS OF OBSERVATION	ERYTHROCYTES (MILLIONS PER CMM)	HEMOGLOBIN (GM PER 100 CC)	ERYTHROBLASTS	LYMPHOID ERYTHROBLASTS	LYMPHOID CELLS	MYELOCYTES	THROMBOCYTES
1	1.48	9.30	1*	0	0	0	2
7	2.46	9.00	1	0	0	0	2
14	2.13	7.45	1	0	0	0	1
21	1.86	6.00	2	1	0	0	2
28	1.98	6.70	2	1	0	0	2
35	.97	2.45	2	2	0	0	2
42	1.66	4.70	3	2	0	0	3
49	1.74	5.65	3	2	1	0	3
56	1.71	5.70	3	1	0	0	3
63	.90	2.65	3	2	1	0	3
70	.87	2.65	3	2	2	0	3
77	1.03	2.05	4	3	1	0	3
84	.68	1.25	4	3	1	1	3
86	.67	1.35	4	3	2	0	3

*The relative increase in the number of pathologic cells observed in the blood smear was graded on a basis of 1 to 4. The relative decrease in number of thrombocytes was also graded on a basis of 1 to 4. 0 indicates that none or very few of the pathologic cells were observed.

TABLE II—*Character of disease produced in chickens experimentally.*

STRAIN	CHICKENS INOCULATED	ERYTHROBLASTIC LEUKOSIS*	GRANULOBLASTIC LEUKOSIS*	INCIPIENT LEUKOSIS†	SPONTANEOUS RECOVERIES	NEGATIVE
I	17	1	2	2	0	12
II	35	16	7	1	2	11
III	121	52	20	5	2	44
Totals	173	69	29	8	4	67

*The designation of the type of disease was dependent on the predominant type of immature cell in the blood at death.
†That type of experimental leukosis in which lesions are limited to the myeloid tissue without the characteristic changes in the peripheral blood.

The type of disease shown by chicken 19 was erythroblastic leukosis. A remission was observed from 42 to 56 days after the onset of symptoms, when the hemoglobin reached a value of 4.70 to 5.70 gm per 100 cc of blood. This chicken died after an observation period of 87 days, and necropsy revealed changes in the organs typical of erythroblastic leukosis.

A male, adult White Leghorn chicken was the source of strain III (chicken 55, fig. 1). This chicken also disclosed the changes in blood and tissue characteristic of erythroblastic leukosis.

Transmission Experiments

The transmission experiments carried out with the various strains are outlined in figure 1. The character of the disease produced in the chicken experimentally are summarized in table II. The incidence of successful transmission of the various strains is given in table III.

Table III—*Successful transmission of various strains.*

Strain	Chickens Inoculated	Leukosis Developed (per cent)	Negative (per cent)
I	17	29.4	70.6
II	35	68.6	31.4
III	121	63.6	36.4
Totals	173	61.3	38.7

Some experiments were carried out for the purpose of comparing certain features of these transmissible strains with strains of the causative agent developed in other laboratories. These experiments involved the filtrability of the etiologic agent and the observation of its resistance to glycerin solution.

In the transmission experiments with strain III, a group of four chickens (141, 142, 143 and 144, fig. 1) were inoculated with a suspension of liver material in glycerin. The inoculum was prepared by grinding a portion of the liver of chicken 131 (which had erythroblastic leukosis) in a 50 per cent glycerin solution. This suspension was kept at ice-box temperatures for 57 days before inoculation. Only one of the four chickens so inoculated developed leukosis. At a later date, two chickens (171 and 172) were inoculated with a mixture of equal parts of glycerin and blood plasma of a chicken with erythroblastic leukosis. This mixture was kept at ice-box temperatures for 20 hours before in-

oculation. One of these two chickens (171) developed erythroblastic leukosis.

Transmission was attempted with filtered blood plasma from chicken 145. The sample of leukemic blood was citrated and centrifuged. The supernatant plasma was pipeted off and mixed with a suspension of *Bacillus prodigiosus*. This mixture was then passed through a Seitz filter. *B. prodigiosus* failed to pass through into the filtrate. The four chickens (153, 154, 155 and 156) which were inoculated with plasma filtrate failed to show changes of leukosis, and three were living eight months after inoculation. One died four months after inoculation with a condition not associated with leukosis. A control group of chickens (149, 150, 151 and 152) were inoculated with whole blood from the same donor. All developed leukosis and died within a period of 25 days after inoculation.

Another attempt to transmit the disease by means of a filtrate was made. A bone-marrow suspension from chicken 157 was passed through a Mandler filter. Culture media inoculated with filtrate failed to grow *B. prodigiosus* which had been added to the suspension. The two chickens (161 and 162) which were inoculated with the filtrate developed granuloblastic and erythroblastic leukosis. One of the two control chickens (163) inoculated with the bone-marrow suspension developed erythroleukosis. The other (164) died with a bacterial infection, the organism of which was similar to *Salmonella gallinarum*.

Two turkeys (5 and 6), two geese (1 and 2) and two ducks (3 and 4) were inoculated with portions of the same filtrate received by chickens 161 and 162. None of these developed leukosis. Two turkeys (7 and 8) and two geese (3 and 4) were inoculated likewise with portions of the bone-marrow emulsion received by chickens 163 and 164. None of these developed leukosis.

With the last two groups of animals inoculated with bone-marrow suspension and filtrate, an attempt was made to demonstrate specific sensitivity to the agent producing leukosis. The antigens were a portion of the same filtrate which was used to inoculate the animals and a suspension of the dried marrow emulsion. The dried marrow emulsion was resuspended so that the dose used to test sensitivity was approximately 2 mg of dried marrow tissue. Intradermal injections of the marrow suspension were made in the left wattle of the chickens and turkeys and on the left side of a plucked area of skin on the neck of the ducks and geese. The filtrate was injected intradermally on the corresponding anatomic locations on the right side. These tests were made

four weeks after the birds had received inoculations of leukosis-producing material. Readings were made of the degree of swelling at the sites of injection five, 24 and 48 hours after injection.

The leukemic chickens (80 and 81) showed marked swelling at the site of injection of both materials at each time of observation. The two turkeys (6 and 7) showed a more marked swelling at the site of injection of the filtered material at each observation. None of the four ducks (1, 2, 3 and 4) or four geese (1, 2, 3 and 4) showed any reaction at the places where the materials were inoculated. A control group of five normal chickens was treated in a similar manner and gave reactions comparable to those observed in the leukemic chickens. Two normal geese also were injected and showed no reaction. It was concluded that no specific sensitivity was demonstrated and that the reactions observed were the result of a nonspecific irritation.

Four turkeys aged four weeks, two ducks aged six weeks, and two chickens (159 and 160) were inoculated with whole blood from chicken 149 which was affected with granuloblastic leukosis. Three of the four turkeys were dead within a week after inoculation. No changes of leukosis were observed. The cause of death of these birds was not apparent. The fourth turkey died five months later from a respiratory disturbance. Duck 2 died shortly after receiving its fourth intravenous inoculation of leukemic whole blood. This bird had typical symptoms of anaphylactic shock after its fourth inoculation, and it was found dead a few hours after apparently having recovered from the shock. No pathologic changes were apparent at necropsy and subsequent histologic examination of the tissues.

Two geese (2 and 3), two ducks (1 and 2) and two turkeys (1 and 7) were each given five inoculations of leukosis-producing material at intervals of a few weeks, in an attempt to produce leukosis. None developed leukosis as a result of this treatment.

Arsenic, in the form of Fowler's solution, was given to one chicken (83, fig. 1) to observe the effect of such treatment. Treatment was begun after the disease had become manifest by changes in the peripheral blood. No demonstrable effect was observed. An attempt was made to saturate two chickens (84 and 86) with arsenic by beginning the course of treatment at the time of inoculation. The chickens died 20 and 50 days later, and neither showed symptoms or changes of leukosis. The death of these chickens may have been due to arsenical poisoning. This

latter type of experiment might be repeated with the use of smaller doses of arsenic.

In several instances experiment birds that were in the advanced stages of the disease were transfused with whole blood of either normal chickens or chickens that had recovered from the disease. The only effect noted was a transient increase in the amount of blood hemoglobin.

Character of the Disease

Morbid anatomy: The gross pathologic changes in the tissues were essentially the same in either erythroblastic or granuloblastic leukosis. In fact, a differential diagnosis between the two could not be made solely on the gross examination of the body of an animal affected with leukosis. The discussion of these changes, therefore, will embrace those of both types of disease.

Almost invariably the chickens would lose weight when affected with this disease. Fowls that developed the disease early in life would remain stunted in their development. The loss of weight was not marked in birds that died shortly after the development of the disease. The blood was pale, watery, and slow to coagulate. An anemic appearance of the viscera was noted in all cases in which the disease was well developed.

Hemorrhage into the tissues was a rather common finding. The mucous membrane of the upper portion of the small intestine was usually the site of considerable hemorrhage. These hemorrhages were not caused by intestinal parasites. Petechial or ecchymotic hemorrhages in the loose areolar subcutaneous tissues were not uncommonly observed. In one chicken there was a massive infiltrative hemorrhage in the tissues in the vicinity of the cloaca. Subcapsular hemorrhage of the liver was observed in a few instances.

Ascites, hydropericardium, and sometimes anasarca, were observed in individuals which had been showing symptoms of the disease over a considerable period of time.

The liver presented rather variable features. In one case (chicken 173) it weighed 114 gm, whereas the entire body weight of the animal was only 460 gm. In other cases the liver was of normal size (40 to 60 gm). Its color varied from yellow or red-brown to purple-brown. Sometimes small discrete white foci were apparent. Recent infarction of portions of the liver were found in a few instances (fig. 2). In these cases the thrombi in the hepatic vessels were not organized.

The spleen was almost always found to be enlarged. This increase in size was variable. In one chicken, which weighed 1,525

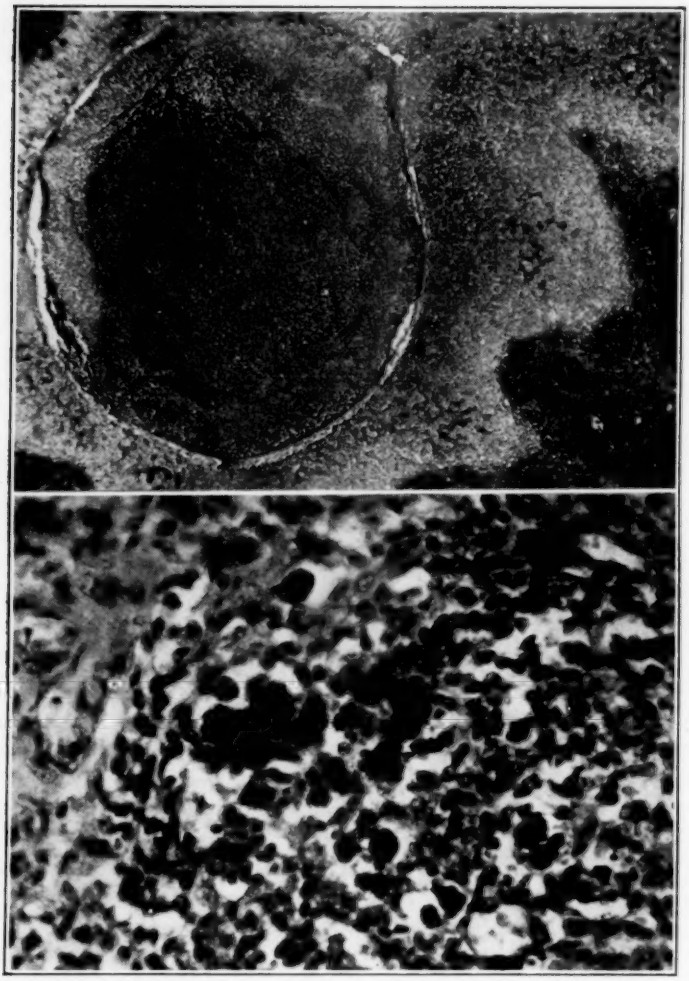

FIG. 2 (above). Thrombosis and infarction in the liver of a chicken (167) affected with transmissible leukosis (hematoxylin and eosin stain, x 40).

Fig. 3 (below). Spleen of a chicken (120) affected with transmissible erythroblastic leukosis. Hemosiderin deposits are present in the reticular cells of the splenic substance (x 525).

gm, the spleen weighed 48 gm (1.5 to 2.5 gm is normal). This organ was usually a deep reddish violet. An increased pressure within the organ was usually demonstrable by the bulging of the cut surface. Usually the organ presented a homogeneous appearance.

The kidneys were enlarged in some instances, although usually they presented no gross changes. The color, as in normal fowls, varied from yellow to a reddish brown.

The thymus glands in some individuals were slightly enlarged and were reddish violet. The lungs and heart showed no gross changes except for their anemic appearance. The ischiatic and brachial nerves were examined routinely and in no instance was there definite pathologic alteration of these structures.

The bone-marrow was hyperplastic. The myeloid tissue contained in the cavity of the femur was examined routinely. The wall of the femur usually was decreased in thickness and the bony lamellae in the marrow cavity were reduced. The myeloid tissue varied from a pink to a reddish purple and was soft in consistency.

The gross changes observed in incipient leukosis were usually confined to the myeloid tissue of the bone-marrow. This type of disease was dependent on the histologic findings for confirmation of the diagnosis.

Histopathology: The fundamental change in fowl leukosis occurred in the myeloid tissue of the bone-marrow. This change appeared to be one in which the blood cells were produced without restriction and the factors which normally retained these cells until they became mature were either overcome or lost, with the resultant outpouring of the immature cells into the circulation.

The microscopic appearance of the bone-marrow in leukosis was that of a tremendous hyperplasia of the myeloid elements. The degree of definitive development in the marrow sinusoids was variable and roughly corresponded in a direct relationship to the number of mature blood-cells seen in the peripheral blood. The marrow in erythroblastic leukosis was principally erythroblastic in character, and in granuloblastic leukosis it was principally granuloblastic. The wall of the femur was thinned, and the bony trabeculae of the medullary cavity were thinned or were completely gone. In some individual cases in which the course of the disease was long, osteoid material was found lining the interior of the wall of the femur. This osteoid material had the appearance of regenerating bony trabeculae.

The microscopic appearance of the liver was variable. Marked stasis of the leukemic cells in the sinusoids often was manifest. In these instances the cords of liver cells were sometimes atrophic and degenerated. This stasis was not marked in some sections of liver studied. The periportal lymphoid tissue was variable in amount. In some instances the lymphoid cells were replaced by accumulations of granulocytic cells. These variable features had been observed also in apparently normal chickens and their significance was not known. Deposits of hemosiderin in the Kupffer cells were frequently observed.

The blood-spaces in the spleen usually were filled to capacity with immature blood-cells. The trabeculae were thinned and their number was reduced. The lymphoid tissue normally present in the malpighian corpuscles was reduced in most instances. Deposits of hemosiderin, whose identity was determined by histologic methods for the identification of iron, were observed sometimes (fig. 3).

The arteriolar bed of the kidney was not infrequently the site of marked stasis of the leukemic cells. In a few instances the immature cells appeared to be undergoing autonomous growth in these areas of stasis. The villi of the intestinal mucosa were frequently engorged with immature blood-cells. The smaller arterioles and capillaries had been ruptured, with resultant hemorrhage into the tissue and intestinal lumen. The thymus gland presented no unusual features other than the intravascular appearance of leukemic cells. The blood vascular bed of the lung in some instances was dilated and filled with immature blood-cells. Histologic examination of the peripheral nerves failed to reveal pathologic alterations of the nerve tissue.

Changes in the blood: Transmissible leukosis is properly considered as a disease of the blood-forming organs and it manifests itself by changes in the cellular composition of the circulating blood. These changes in the blood-cells were quite variable. The qualitative changes that occurred were of chief interest. Certain quantitative changes also were present but these varied with the individual cases and are of less significance.

In this study the pathologic blood-cells were classified in groups* as follows:

Group 1 consisted of erythroblasts, which group included all hemoglobin-bearing cells. In leukosis the mature erythrocyte assumed many bizarre forms. Poikilocytosis (variations in shape), anisocytosis (variations in size) and polychromasia

*Furth[4] and Oberling and Guérin[5] show colored plates which portray these cells.

(variations in color of the cytoplasm) were features presented by the erythrocytes. Occasionally binucleated erythrocytes were observed, and not infrequently a red blood-cell without a nucleus was seen in leukotic blood. Polychrome erythrocytes were regarded as those erythrocytes which did not have their full quota of hemoglobin in the cytoplasm. They were usually more spherical than oval. The basophilic zones of cytoplasm near the cell margin surrounded a well-marked zone of hemoglobin-containing cytoplasm immediately about the nucleus. The nucleus was usually somewhat larger than that observed in the normal erythrocyte. This cell was not uncommonly seen in otherwise normal blood. The polychrome erythroblast was larger than the polychrome erythrocyte and represented a less mature form of the red-cell series. The basophilic margin was of a deeper tint and much broader. The perinuclear zone of hemoglobin was narrow and often only faintly discernible. The nucleus was relatively large and had the features of being less mature when contrasted with those of the polychrome erythrocyte. Binucleated forms of these cells often were seen in leukotic blood.

Group 2 consisted of lymphoid erythroblasts which were considered as a form of immature blood-cell that had the features of both their precursors, the lymphoid cells, and their successors, the polychrome erythroblasts. These cells were large, usually round, and often had a slightly irregular outline. The cytoplasm was narrow and basophilic, appearing to be finely granular. Usually a slightly lighter-staining perinuclear zone was evident. Mitotic figures often were seen. Occasionally binucleated forms were observed. Vacuolization of the cytoplasm was a peculiar characteristic, which was observed often. The chromatin and parachromatin of the nucleus were rather sharply delineated and were arranged in a fine pattern.

Group 3 consisted of myelocytes which were considered as including all the young granulocytes as well as their precursors, the leukoblasts. Granulocytes of varying stages of development were observed.* Leukoblasts were recognized as a type of cell in which the specific granulation had not yet made its appearance. Nucleoli were not present. The nucleus was made up with a fine chromatin and parachromatin arrangement. The cytoplasm was blue-gray. Variable features of this cell often were observed. The nucleus was broken up into two or three lobes which were connected by thin strands of chromatin. These cells presented features which made them analogous to Rieder cells, that

*Oberling and Guérin[5] show a colored plate in which the myelocytes are well portrayed.

is, cells having a cytoplasm like the leukoblast but a more mature form of nucleus (fig. 4). Large granules of pre-acidophilic basophilic material were observed in many metamyelocytes. These basophilic granules were of variable size. Some cells were observed in which granules of varying shades of basophilic to faint acidophilic staining were apparent. One blood smear was examined in which many metamyelocytes contained large faintly acidophilic bodies (fig. 5). The size and general shape of these bodies were reminiscent of the appearance of Russell's fuchsin bodies of plasma cells stained by Dominici's technic. The exact nature of these bodies or their significance is not known. They probably represent an abnormal formation of the specific granu-

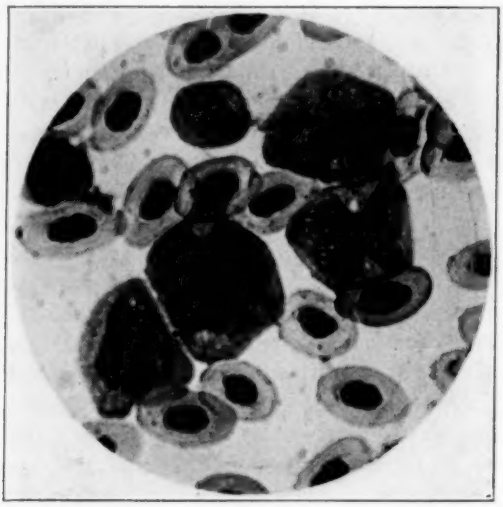

FIG. 4. Blood smear of a chicken (36) affected with transmissible granuloblastic leukosis of the Rieder-cell type. The large immature cells which show the tendency toward lobulation of the nucleus may be noted (Wright-Giemsa stain, x 1,200).

lation. When the development of the granulocytic blood-cell was affected markedly by the inciting agent of transmissible leukosis, the majority of the immature cells in the circulating blood were metamyelocytes and leukoblasts. The myelocytes and granulocytes were not numerous.

Group 4 consisted of lymphoid cells, which were recognized by the very fine chromatin and perichromatin arrangement of the nucleus and by the blue-gray basophilic cytoplasm. Nucleoli were observed sometimes. These cells were usually larger than the

lymphoid erythroblasts. Occasionally, vacuoles were observed in the cytoplasm.

The thrombocytes also were involved in the fowl leukosis. Their number was reduced early in the disease and atypical forms were observed in the blood.* The cytoplasm was either reduced or was increased in amount. Vacuoles were sometimes seen in the cells.

Actual counts were not made of the immature blood-cells in this study. The relative number of the cells observed in the blood-smear obtained at the time of examination of the blood was graded on the basis 1 to 4. The relative increase of erythroblasts, lymphoid erythroblasts, lymphoid cells, and myelocytes was

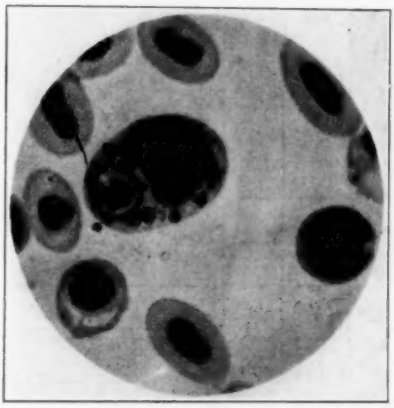

FIG. 5. The largest cell in the field is a promyelocyte, which shows preacidophilic basophilic granulation. The single large granule in the cytoplasm represented a peculiar finding. Its reaction to Wright's stain was reddish-brown. It has a rough appearance. The significance of this mass of material is not known, but it was probably the result of the abnormal metabolism of the leukemic cell (Wright's stain, x 1,200).

graded according to the following criteria: grade 1, slightly more than observed in normal blood; grade 2, several; grade 3, many, and grade 4, abundant. The relative diminution of thrombocytes also was classified on the basis of 1 to 4 as follows: grade 1, a few less than normal; grade 2, definite diminution; grade 3, very marked decrease, and grade 4, very few present.

The changes in the blood appeared after an incubation period of variable duration. This incubation period might be short,

*Oberling and Guérin[5] show some of the atypical forms of these cells in their colored plates.

as was the case in chickens 42 and 43 (fig. 6) and 28 (fig. 7), which consisted of 22, 11, and 14 days, respectively. The longest incubation period observed was 172 days, for chicken 32 (fig. 8).

Usually the first change observed was a decrease in the number of thrombocytes, which was closely followed by a drop in the number of erythrocytes and in the amount of hemoglobin and by the appearance of immature cells. This was well illustrated by chickens 20, 28 and 31 (fig. 7). The decrease in throm-

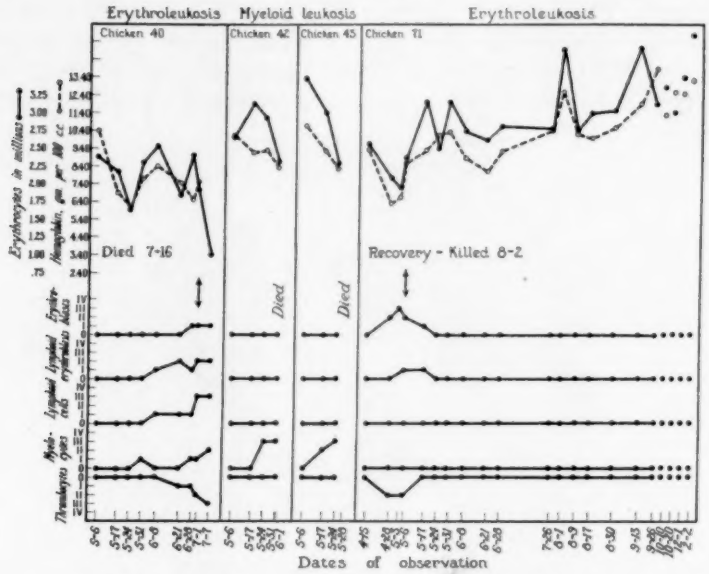

FIG. 6. Observations on the blood of chickens (40, 42, 43 and 71) affected with transmissible leukosis. Chicken 40 had only a slight anemia when many immature blood cells were manifest; chickens 42 and 43 had a severe form of granuloblastic leukosis. Chicken 71 recovered spontaneously from erythroblastic leukosis. These chickens were inoculated at the time of the first blood examination recorded. The arrows indicate that on that date material was obtained from the fowl for further animal passage. These results are shown in fig. 1.

bocytes might, however, not become apparent until after the other features were manifest (chicken 40, fig. 6). The number of erythrocytes and the amount of hemoglobin might actually increase in the beginning of the disease, as was observed in chicken 25 (fig. 9).

In one chicken (62, fig. 10), pathologic cells were not observed in the blood in the course of the disease, with the exception of one examination. At this time, 98 days after inoculation, a few polychrome erythrocytes were apparent. The number of erythrocytes and the amount of hemoglobin may have been reduced

slightly, but certainly not to the extremely low levels usually observed. The chicken died 109 days after inoculation. Microscopic examination of the liver failed to reveal any change in the blood in the larger blood-vessels. The bone-marrow tissue, however, did reveal changes that could be attributed to the action of the transmissible agent. It was also of interest that blood obtained from this chicken, 94 days after inoculation, produced leukosis in four fowls, into which it was inoculated intravenously.

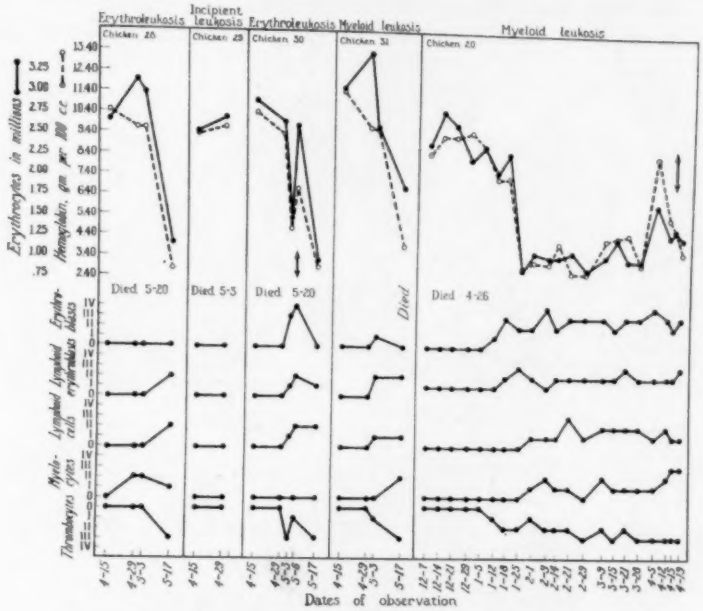

FIG. 7. Observations on the blood of chickens (28, 29, 30, 31 and 20) affected with transmissible leukosis. Chickens are inoculated on the first date on which the blood examination is recorded. Arrows indicate that on that date material was obtained for transmission experiments.

With chicken 61 (fig. 11) there was a peculiar incubation period. Sixty-six days after inoculation the blood-smear revealed a decrease in the thrombocytes and a slight increase in the erythroblasts. For the next 57 days these were variable in number. During all this time the number of erythrocytes and the values for hemoglobin were slowly dropping. One hundred sixty-eight days after inoculation, a remission occurred and lasted for 43 days, after which changes in the blood were observed frequently until the chicken's death, 552 days after inoculation. This record is unique in that leukosis was of longer duration in this instance than in any case reported heretofore. During the

course of this chicken's illness, there were six instances (80, 94, 108, 115, 192 and 198 days, respectively, after inoculation) when examinations of the blood made after the beginning of the disease showed no pathologic changes in the cells.

Complete recovery was observed in four instances (chickens 22, 45, 59 and 71, figs. 1, 6 and 10). Chicken 22 showed changes in the blood typical of erythroblastic leukosis during the period between 22 and 42 days after inoculation. During this time the number of erythrocytes and the amount of hemoglobin also were reduced. From the time of the examina-

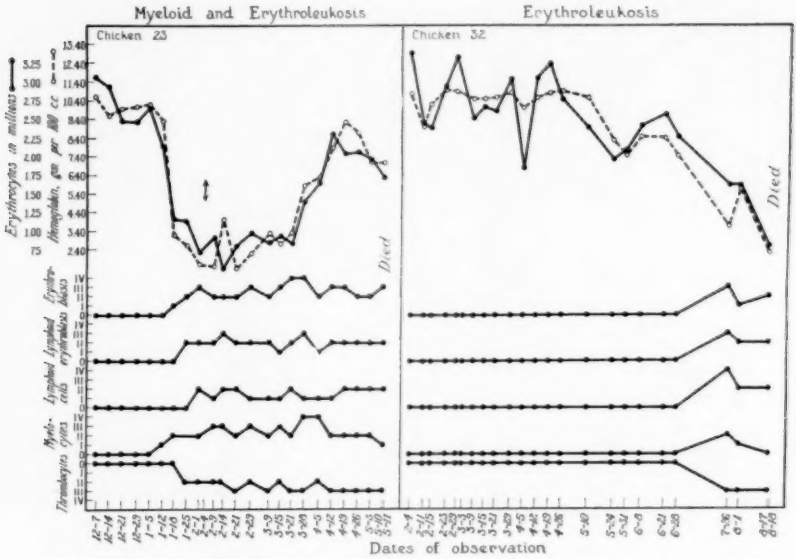

FIG. 8. Observations on the blood of chickens (23 and 32) affected with transmissible leukosis. The arrow indicates that material was obtained on that date for further transmission experiments. The birds were inoculated on first date shown. In the case of chicken 23, many immature cells of both the erythroblastic and granuloblastic series were present in the peripheral blood. In the case of chicken 32, the inoculation period was long.

tion, made 49 days after inoculation, until the chicken was killed 449 days later, there were no changes in the blood which could be attributed to leukosis. Erythroblastic leukosis developed in chicken 45, 53 days after inoculation, and it continued to become progressively worse. Examination of the blood, 34 days after the onset of the changes in the blood, revealed only 660,000 erythrocytes per cmm and 2.35 gm of hemoglobin per 100 cc of blood. Examination of the blood, 132 days after inoculation, was negative, as were the examinations made thereafter. The chicken was

killed 690 days after inoculation, at which time no evidence of leukosis was apparent.

Chicken 71 (fig. 1) was another example of spontaneous recovery following leukosis. This chicken served as donor for the transmission of the disease to another group of chickens and demonstrated that the inciting agent was present and viable in the blood of such fowls during the time the changes in the blood were observed. The first changes in the blood were noted in chicken 71, 13 days after inoculation, and persisted over a

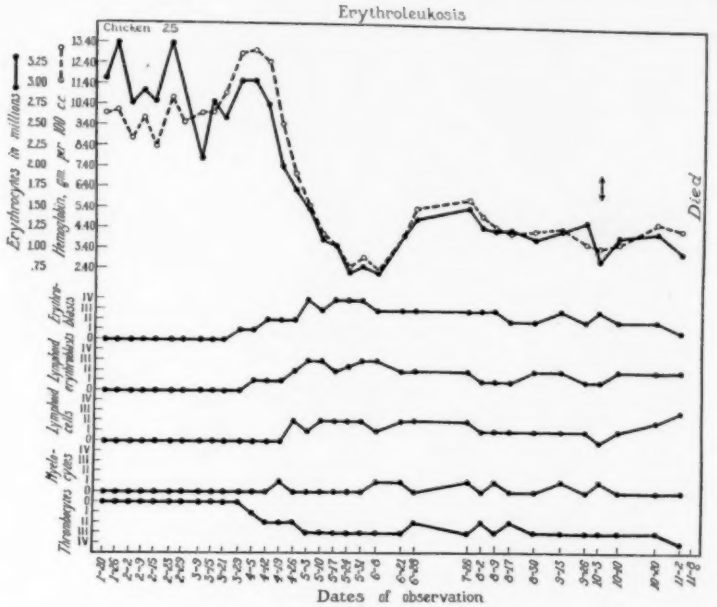

FIG. 9. Observations on the blood of a chicken (25) inoculated (1 to 20) with whole blood from chicken 19. Note the rise in hemoglobin and erythrocyte values before the usually observed drop of these values. On October 3, whole blood from this fowl was used to inoculate other animals.

period of 19 days, after which time they were no longer apparent. This chicken died 474 days after inoculation. No lesions of leukosis were demonstrated at necropsy, death being attributed to nutritional disturbances.

Some chickens died within a relatively short time after receiving the infective material. Examples of this type of reaction were shown by chickens 28, 29, 30 and 31 (fig. 7), and by 42 and 43 (fig. 6). The changes in the blood, when observed, were either of granuloblastic or erythroblastic leukosis. In some in-

stances the experimental chicken died without showing changes in the peripheral blood (chicken 29, fig. 7).

Most fowls showing the changes in the blood of erythroblastic leukosis, when the erythrocytic series of blood-cells was affected, also showed immaturity and pathologic changes of the granuloblastic series. This may be noted in figure 6 (chicken 40), figure 11 (chicken 61); and in figure 9 (chicken 25).

In some instances severe granuloblastic leukosis was observed, in which no pathologic changes in the erythrocytes were apparent (chickens 42 and 43, fig. 6). The circulating blood became so

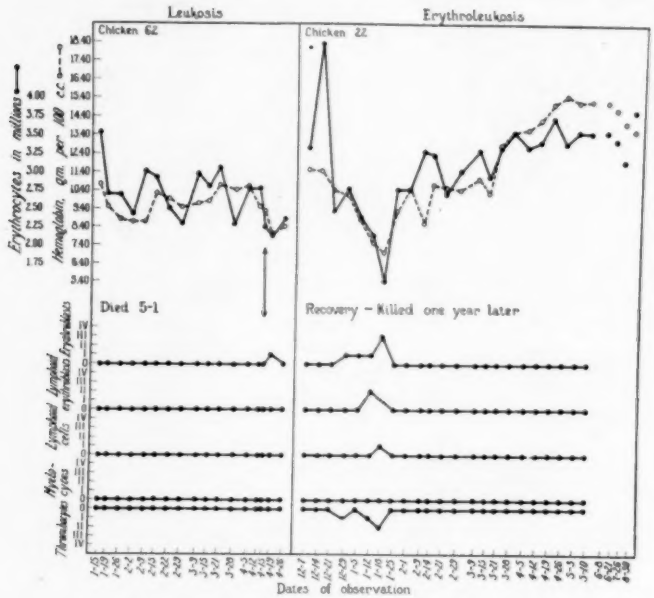

FIG. 10. Observations on the blood of chickens (62 and 22) affected with transmissible leukosis. The chickens were inoculated at the time of the first recorded blood examination. Material from chicken 62, obtained April 15, produced the disease in three or four chickens inoculated. The fact will be noted that only minor pathologic changes were shown in the blood during the course of the chicken's illness. Chicken 22 recovered from the disease.

overwhelmed with immature granuloblastic cells that it was difficult to recognize changes in the other blood-cells. Often, in these cases, the bulk of the immature cells were atypical leukoblasts similar to Rieder cells.

The changes in the blood observed during the course of the disease in certain cases were so variable that, at times, the term "erythroblastic" leukosis was applicable and at others the term

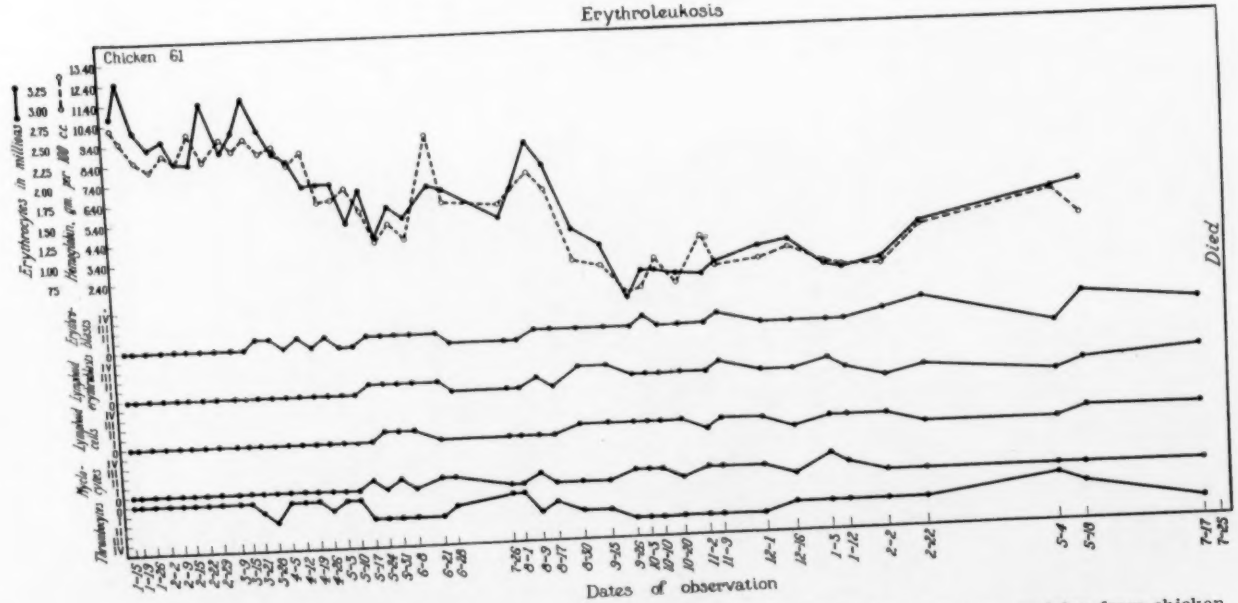

FIG. 11. Observations on the blood of a chicken (61) inoculated with a bone-marrow emulsion from chicken 55 (spontaneous erythroleukosis) January 15. The extreme length of the period during which time the changes in the blood were very variable will be noted.

"granuloblastic" leukosis was suitable. Chicken 20 (fig. 7) and chicken 23 (fig. 8) were examples of this type of reaction.

The study of the many variable features that were observed in the blood led to the conclusion that it was rather pointless to distinguish between erythroblastic leukosis and granuloblastic leukosis. Transitional forms in the blood in both diseases occurred not only in individual fowls, but also during the course of the disease in a single fowl.

Comparison with spontaneous leukosis: Several cases of spontaneous leukosis were observed among the chickens comprising the stock animals. From many of these, blood-smears were made just before death or at the time of necropsy. There were no essential features of the spontaneous form of fowl leukosis which differed from those observed in the experimentally produced lesions.

Summary and Comment

None of the three experimental strains revealed a tendency to produce only one type of leukosis. The disease was transmitted to the majority of the inoculated birds. The more common type of the disease was erythroblastic leukosis.

In general, the infecting agents were more effective in young fowls than in older fowls. It was also generally true that chickens of the Barred Plymouth Rock breed were somewhat more susceptible than the other breeds used. This higher degree of susceptibility was shown by the higher incidence of successful inoculations, and the more acute form of disease developed in the Barred Plymouth Rock chickens.

The gross and microscopic pathologic changes observed were similar to those described by certain other investigators and were characteristic of transmissible leukosis. There were, however, no neoplastic conditions of endothelioma or sarcoma observed in the experimental animals as described by Furth,[6, 7] Rothe Meyer and Engelbreth-Holm,[8] and Oberling and Guérin[9, 10] in certain of their transmissible strains of fowl leukosis.

No instances of neurolymphomatosis gallinarum (fowl paralysis) were observed in the experiment birds, although a careful search was made to ascertain the presence of this condition. Only two instances of lymphocytoma were observed among the 173 experiment birds. This extremely low incidence is remarkable in some respects, as the spontaneous occurrence of this disease has been reported to be much higher. These facts indicated that, at least in these three strains of the transmissible agent, there was no etiologic relationship between transmissible

leukosis, lymphocytoma and neurolymphomatosis, as has been maintained by certain investigators, among whom are Patterson and his associates,[11] and Johnson.[12]

The changes in the blood were found to be very variable in the course of the experimental form of the disease. Deviations of the blood from normal were observed in practically all cases of transmissible leukosis. Certain cases, such as those of incipient leukosis, showed only minor or no pathologic changes. In some instances the changes were marked and a state of severe anemia was manifest (300,000 erythrocytes per cmm and 0.80 gm of hemoglobin per 100 cc of blood). In other instances the changes in the blood were negligible, yet the blood did contain the inciting agent of transmissible leukosis (chicken 62).

Periods of remissions and exacerbations of the blood symptoms were observed in the course of the disease. The changes in the blood were discussed in detail in the section devoted to the subject. Several authors have discussed the similarity of transmissible leukosis to certain dyscrasias of the blood in man.

Ellermann[13] believed erythroblastic leukosis to be analogous to pernicious anamia. Bayon[14] expressed a similar viewpoint. The basis of these opinions is chiefly the very severe degree of anemia observed in both diseases. There are, however, many features in which the diseases are dissimilar. Combined degenerative sclerosis of the spinal cord and atrophy of the gastric mucosa are not constantly, if ever, observed in fowl leukosis. Henschen[15] and Jármai[16] pointed out several features in common between erythroblastic leukosis and the disease, anemia pseudoleukemia infantum. Furth[4] and Engelbreth-Holm and Rothe Meyer[17] compared erythroblastic leukosis with erythroblastosis congenita.

Granuloblastic leukosis of chickens is similar to myelogenous leukemia of man in many respects. Fundamentally, however, transmissible leukosis has no counterpart in any other species of animal. The close relationship, if not the almost exact identity, of erythroblastic and granuloblastic transmissible leukosis, indicates that they are responses to the same irritant. No such relationship is known to exist in the hemopathies of man or of other animals.

Mention should be made also of the nomenclature of this disease. The term "myeloid" leukosis was introduced by Ellermann and has been applied since by most individuals to indicate the type of leukosis in which the major disturbance is of granulocytopoiesis. The distinction of "erythroleukosis" from "myeloid leukosis" also has been made. The myeloid tissue of the fowl is the site of both erythropoiesis and granulopoiesis. It there-

fore seems that "myeloid" leukosis is rather an ill chosen term when it is intended to refer specifically to "granuloblastic" leukosis. From a practical standpoint it is not essential to distinguish between the erythroblastic or granuloblastic forms of the disease. The term "fowl leukosis" has been rather loosely applied to certain conditions not produced by the etiologic agent of transmissible leukosis. The term "transmissible fowl leukosis" is suggested for this disease. When necessary, this term may be further modified by the word "erythroblastic" or "granuloblastic," as for example, "transmissible erythroblastic leukosis."

Of 173 chickens that were inoculated in transmission experiments with three strains of inciting agents of transmissible leukosis, the erythroblastic form developed in 69 (four of which recovered spontaneously), the granuloblastic form developed in 29, eight died showing changes of incipient leukosis, and 67 remained negative. The inoculums used in the transmission experiments were whole blood, blood plasma, or emulsions of liver or bone-marrow.

Studies of the filtrability of the transmissible agent were made with the use of Seitz and Mandler filters. The viability of the agent in 50 per cent glycerin solution was studied.

Attempts were made to produce the disease in ducks, geese and turkeys by repeated inoculations. Certain experiments were conducted in an attempt to demonstrate intradermal sensitivity of these animals to the inciting agent of transmissible leukosis; the results were essentially negative.

Observations were made on the gross and microscopic, organ and tissue changes of the disease. The changes in the blood in the experimental form of the disease were studied in detail and the variable features were noted. The pathologic changes in the experimentally produced disease were compared with that observed in several spontaneous cases of fowl leukosis.

Conclusions

1. Fowl leukosis is transmissible to susceptible chickens by means of whole blood, blood plasma, and tissue emulsions.

2. The inciting agent remains viable in glycerin solution and is filtrable.

3. The disease is not transmissible to ducks, geese or turkeys.

4. The disease produced by the inciting agent is manifest by certain specific pathologic changes in the erythrocytic and granulocytic hematopoietic tissues, and in most instances by the appearance of pathologic cells in the peripheral blood.

5. The transmissible agent of fowl leukosis is not responsible for the conditions known as lymphocytoma or neurolymphomatosis gallinarum.

6. The changes in the blood in transmissible fowl leukosis are variable quantitatively and qualitatively.

7. Transfusions with chicken blood and oral administration of arsenic in the form of Fowler's solution are of no avail in the treatment of the well-established form of the disease.

REFERENCES

[1]Wiseman, B. K.: An improved direct method for obtaining the total white cell count in avian blood. Proc. Soc. Exp. Biol. & Med., xxviii (1931), pp. 1030-1038.

[2]Olson, C. Jr.: Available methods for examination of the blood of the fowl. Jour. A.V.M.A., lxxxvi (1935), n.s. 39 (4), pp. 474-487.

[3]Slider, E. M., and Downey, H.: Methods for the study of leukocytes. In: McClung, C. E.: Handbook of Microscopic Technic. pp. 243-256. (Paul B. Hoeber, New York, 1929.)

[4]Furth, J.: Erythroleukosis and the anemias of the fowl. Arch. Path., xii (1931), pp. 1-30.

[5]Oberling, C., and Guérin, M.: La leucémie érythroblastique ou erythroblastose transmissible des poules. Bul. de l'Asso. franc. p. l'étude du cancer, xxiii (1934), pp. 38-82.

[6]Furth, J.: Lymphomatosis, myelomatosis, and endothelioma of chickens caused by a filterable agent. I. Transmission experiments. Jour. Exp. Med., lviii (1933), pp. 253-275.

[7]Furth, J.: Lymphomatosis, myelomatosis, and endotheliomata of chickens caused by a filterable agent. II. Morphological characteristics of the endotheliomata caused by the agent. Jour. Exp. Med., lix (1934), pp. 501-517.

[8]Rothe Meyer, A., and Engelbreth-Holm, J.: Experimentelle Studien über die Beziehungen zwischen Hühnerleukose und Sarkom an der Hand eines Stammes von übertragbarer Leukose-Sarkom-Kombination. Acta. path. et microbiol. Scandin., x (1933), pp. 380-428.

[9]Oberling, C., and Guérin, M.: Lésions tumorales en rapport avec la leucémie transmissible des poules. Bul. de l'Asso. franc. p. l'étude du cancer, xxii (1933), pp. 180-212.

[10]Oberling, C., and Guérin, M.: Nouvelles recherches sur la production de tumeurs malignes avec le virus de la leucémie transmissible de poules. Bul. de l'Asso. franc. p. l'étude du cancer, xxii (1933), pp. 326-360.

[11]Patterson, F. D., Wilcke, H. L., Murray, C., and Henderson, E. W.: So-called range paralysis of the chicken. Jour. A.V.M.A., lxxxi (1932), n.s. 34 (6), pp. 747-767.

[12]Johnson, E. P.: The etiology and histogenesis of leucosis and lymphomatosis of fowls. Va. Agr. Exp. Sta. Tech. Bul. 56 (1934).

[13]Ellermann, V.: Histogenese der übertragbaren Hühnerleukose. II. Die intravaskuläre lymphoide Leukose. Folia haematol., xxvi (1921), pp. 165-175.

[14]Bayon, H. P.: The pathology of transmissible anaemia (erythromyelosis) in the fowl: its similarity to human haemopathies. Parasitol., xxi (1929), pp. 339-374.

[15]Henschen, F.: Quoted by Jármai.

[16]Jármai, K.: Die Leukosen der Haustiere. Ergeb. d. all. Path. u. path. Anat., xxviii (1934), pp. 227-312.

[17]Engelbreth-Holm, J., and Rothe Meyer, A.: Bericht über neue Erfahrungen mit einem Stamm Hühner-erythroleukose. Acta. path. et microbiol. Scandin., ix (1932), pp. 293-312.

Animal Products in Ford Cars

One of Henry Ford's statisticians has figured out that a million Ford V8's require 3,200,000 pounds of wool, from 800,000 sheep; leather, glue, grease and glycerin from 30,000 cattle; oil and bristles from 20,000 hogs; 350,000 pounds of goat hair (for mohair), from 87,500 goats, and beeswax from 93,000,000 bees.

BETTER GENERAL ANESTHESIA IN ANIMALS

II. Comparative Study of Morphine, Pentobarbital Sodium (Nembutal), and Sodium Amytal as Pre-Anesthesia Sedatives and Hypnotics*

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There are certain definite advantages in the use of appropriate pre-anesthesia sedative and hypnotic doses of medicinal agents. Of the various chemical substances available, perhaps morphine and the short-acting barbiturates are of more use in small-animal practice. In the large animals there has been a greater difficulty to find agents that are suitable, and yet comparatively non-toxic. Chloral hydrate and some of the barbiturates have been considered.

For a long time, morphine alone, or in combination with other agents (H.M.C., etc.), was found a useful and relatively non-toxic pre-anesthesia sedative for the dog. In recent years the short-acting barbiturates, particularly pentobarbital sodium, or nembutal (mono-sodium salt of ethyl methylbutyl barbituric acid) have been introduced, not only as sedative and hypnotic agents, but also for their narcotic action, to take the place of the general anesthetics in minor and even major surgical operations. Some of the barbiturates have been in use in human medicine in sedative and hypnotic doses (sodium amytal-ethyl-isoamyl-barbituric acid, etc.) as basal anesthetics supplemented by small doses of general anesthetics, in which ether-oxygen or nitrous oxide-oxygen was used. Anderson,[1] Chen and Leake have indicated that barbiturates might be substituted for morphine pre-anesthetically. The primary object was to spare the patient the psychic shock during induction, and to obtain more even general anesthesia. Better relaxation and smoother recovery was possible with less ether, thus simplifying the problem of post-anesthesia nursing.

In view of the fact that morphine produces nausea in the dog,[2] and in some cases vomiting, etc., particularly when the patient is not prepared for anesthesia, it was thought desirable to make a comparative study of the short-acting barbiturates and morphine as pre-anesthesia sedatives, to learn if supplemental

*Received for publication, June 29, 1936.

agents such as the barbiturate group could be introduced as convenient pre-anesthesia sedative in animal surgery.

The Method and Apparatus Used

To determine the comparative pre-anesthesia sedative influence of morphine, pentobarbital sodium (nembutal), and sodium amytal, the dogs used in experimentation were given a sedative dose three times, and each dose was followed by general anesthesia. Morphine was used first, then pentobarbital sodium, and finally sodium amytal.

Thus, each animal was anesthetized three times, and a complete record was obtained pre-anesthetically, during anesthesia, and post-anesthetically, when the animal recovered. Sedative doses of the drugs were injected subcutaneously under sterile conditions. Intravenous injections were not used, since in animal practice the subcutaneous route is more practical and convenient, thus saving time for the practitioner. Furthermore, it simplifies the mode of handling; that is, the animal need not be restrained. The dosage of the barbiturates was based on the body weight and calculated in 1/10 gr. per pound of body weight. Other doses were used (the narcotic dose as recommended is 1/5 gr. per pound body weight), such as ⅛ gr. per pound of weight, but finally 1/10 gr. was adopted. This dose produced sufficient sedative action to reduce struggling and cause some relaxation preparatory to anesthesia, but without hypnotic effect. The morphine was used in doses of from ¼ to ½ gr., depending upon the size of the animal, and its probable susceptibility.

The interval elapsing between the time of administration of the sedative dose and the time of induction of general anesthesia was, on the average, about 15 minutes. In a few cases it was necessary to allow 20 minutes for the particular barbiturate to be absorbed sufficiently to cause relaxation and quiet the animal. The mode of induction and the apparatus used were based upon the previous work of Covington,[2] in which the ether in oxygen was dispensed in a form of vapor by use of an appropriate apparatus (closed method) through a special inhaler mask. The apparatus was somewhat modified, however, in that better vaporization and control of the flow of gas was made possible. The animals, all of which were pound dogs, were not prepared for the anesthesia.

Comparative Results

Twenty dogs were anesthetized. Fifteen of them received anesthesia three times, with an appropriate pre-anesthesia drug.

The five remaining dogs received anesthesia only two times. With each anesthesia, a different pre-anesthesia sedative was used.

In table I the comparative average induction time, the recovery time, and the amount of ether used are indicated for ten dogs, representing 30 anesthesias maintained for a period of 30 minutes.

TABLE I—*Thirty-minute anesthesia in 10 dogs (average results of 30 anesthesias).*

	Pre-Anesthesia Drug Used		
	Morphine	Pentobarbital Sodium	Sodium Amytal
Induction time (minutes).....	1.25	2.50	2.20
Recovery time (minutes).....	33.20	66.30	26.20
Ether used (cc).....	56.65	66.30	59.90

From the table it may be seen that with barbiturates there was a slight increase in the induction time, and more ether was required. The postanesthesia recovery time was increased with pentobarbital sodium, but it was reduced with sodium amytal, as compared with the morphine. The relative difference in the action of the barbiturates and morphine alone is primarily in the postanesthesia recovery time from pentobarbital sodium. During anesthesia the barbiturate dogs required more constant flow of the anesthetic to maintain surgical anesthesia. The anesthesia was not so smooth as with morphine; respirations were more shallow and the pulse faster.

The most objectionable difference was found in the behavior of the animals during the postanesthesia recovery period. Without exception, all barbiturate dogs were restless postanesthetically, and also during the recovery phase from the anesthesia. Some of the dogs were so greatly excited, that it was difficult to control them, several requiring forcible restraint. In fact, two of the dogs were so violent from amytal, that one of them was given a dose of morphine to cause relaxation. A similar condition in dogs was observed by Wright.[3] In the human the susceptibility is just as great. In some cases the excitement was sufficiently violent to require prompt administration of morphine, constant vigilance, and special nursing. McKesson and McCarthy[4] had found restlessness in only 3 per cent of patients from the use of pernocton (sodium salt of secondary butyl-beta-bromallyl

barbituric acid), as compared to sodium amytal patients, half of whom showed restlessness and several required forcible restraint.

Struggling, restlessness and excitement are not desirable after a major operation; furthermore, in animal surgery, special nursing or restraint cannot be maintained without increasing the cost of medication and care. This is decidedly a disadvantage from the standpoint of the short-acting barbiturates when used pre-anesthetically. The recovery from morphine is smooth, without struggle or excitement. The animal is usually relaxed after recovery, and remains drowsy for a time. Combinations of morphine and one of the short-acting barbitals might be used pre-anesthetically; it was not so used, however, in this problem. It is questionable if such a combination is of value when general anesthesia is to be administered.

There are certain other objections to the use of barbiturates pre-anesthetically, particularly when hypnotic and narcotic doses are administered. More rapid fall in the blood pressure is quite constant. This is most noticeable when larger doses are administered. Decreased cardiac tonus, vaso-dilation of the peripheral vessels, depression of respiration, inconstant action of the adopted doses, and the inconstant onset of sedative, hypnotic and narcotic action, are some of the disadvantages. Used experimentally, the recommended doses, and other doses of barbiturates, did not act the same in all animals. Therefore, definitely standardized pre-anesthetic doses cannot be worked out.

Barbiturates, however, do fill a useful place in animal medicine, even though not all animals respond identically to a given dose. In minor surgical operations, or in the handling and examining of difficult or vicious animals, and in many other conditions where sedative and hypnotic influence of the drug is desired, short-acting barbiturates are indicated.

SUMMARY

1. Doses of pentobarbital sodium (nembutal) and sodium amytal were administered to animals pre-anesthetically, to find a suitable sedative agent to take the place of morphine.

2. Objectionable restlessness and struggling were noted during the recovery from ether-oxygen anesthesia, when barbiturates were used. Doses of 1/5 gr., 1/8 gr., and 1/10 gr. per pound of body weight were used experimentally. The 1/10 gr. per pound weight was adopted and found best. The inconstant response of animals to a given dose of barbitals made it difficult to standardize definitely a pre-anesthetic dose.

REFERENCES

[1]Anderson, H. H., Chen, M. Y., and Leake, C. D.: Possible substitutes for morphine in pre-anesthesia medication: Observation of the common barbituric acid derivatives. Jour. Phar. & Exp. Therap., xxxix (1930), p. 271.

[2]Covington, N. G.: Better general anesthesia in animals. I. Oxygen-ether controlled anesthesia by the closed method in the dog, with morphine as pre-anesthesia hypnotic. Jour. A.V.M.A., lxxxvii (1935), n. s. 40 (5), pp. 562-572.

[3]Wright, J. G.: Nembutal narcosis in the dog and cat. Proc. Roy. Soc. Med., xxvi (1933), pp. 26-29.

[4]McKesson, E. I., and McCarthy, K. C.: A comparison of sodium amytal and pernocton as pre-anesthesia hypnotics. Read before Section on Anesthesia, Brit. Med. Asso., Winnipeg, Canada, August 29, 1930.

A Staggering Figure

Did you receive a copy of *The Bloodless Phlebotomist* (Vol. VIII, No. IV) just off the press? There were 1,450,000 copies printed. A small publication of only 24 pages, yet it carries between its covers a variety of articles which veterinarians will find of interest.

Among the non-technical articles is one which is, appropriately enough, entitled "Staggering." It is the story of a native of Bombay who claims descent from one of the "Wise Men of the East" of biblical fame, and from which one learns the amazing fact that every living person on earth has had, since the birth of Christ alone, the prodigious number of 144,115,188,075,855,870 ancestors, and the article tells how this figure is, simply enough, arrived at.

If you failed to receive a copy of this interesting publication, one will be sent you if you write The Denver Chemical Manufacturing Company, 163 Varick St., New York, N. Y.

Publications Received

Fertility in Sheep. Artificial production of seminal ejaculation and the characters of the spermatozoa contained therein. R. M. C. Gunn. (Bul. 94, Coun. Sci. & Ind. Res., Commonwealth of Australia, Melbourne, 1936. pp. 116. Illus.)

Observations of *Myxomatosis cuniculi* (Sanarelli) Made with a View to the Use of the Virus in the Control of Rabbit Plagues. Charles J. Martin. (Bul. 96, Coun. Sci. & Ind. Res., Commonwealth of Australia, Melbourne, 1936. pp. 28. Illus.)

Spirochaeta from Blood and Tissue Cultures of Diseased Chickens. Edward Redowitz. Reprint from *Amer. Jour. Med. Tech.*, ii (1936), 3, pp. 92-97. Illus.)

Studies in Brucella Infections. I. Forest Huddleson, J. W. Scales, O. J. Sorenson, A. D. Hershey, H. W. Johnson, D. B. Meyer and Colin P. Beattie. (Tech. Bul. 149. Mich. Agr. Exp. Sta., May, 1936. pp. 51.)

Pathology of Rickets in Dairy Calves. H. Ernest Bechtel, E. T. Hallman, C. F. Huffman and C. W. Duncan. (Tech. Bul. 150. Mich. Agr. Exp. Sta., May, 1936. pp. 47. Illus.)

MULTIPLE PAPILLOMATA OF THE ESOPHAGUS IN A BOVINE*

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Papillomas are of common occurrence in the bovine and are usually confined to the cutaneous surface. In the majority of

FIG. 1. Multiple papillomata of the esophagus of a cow.

cases, the growths are multiple, appearing on the integument over the head, neck, shoulders, teats, and on the limbs. Although the cutaneous surfaces show more frequent involvement,

*Received for publication, September 21, 1936.

the mucous membranes also may be affected. This is particularly true in dogs, where buccal papillomata are frequently observed. In the bovine, however, papillomas of the mucous membrane are less often seen. Feldman[1] states that papillomas may arise in the mucosa of the esophagus, but has not encountered such a case in his study of neoplasms. He did, however, see a papilloma removed from the rumen of a cow. Lisi[2] reported a papilloma of the urethra and bladder of a cow. In view of the few recorded cases of papillomata of the mucous membrane in the bovine, the writer feels that such a case is worthy of report.

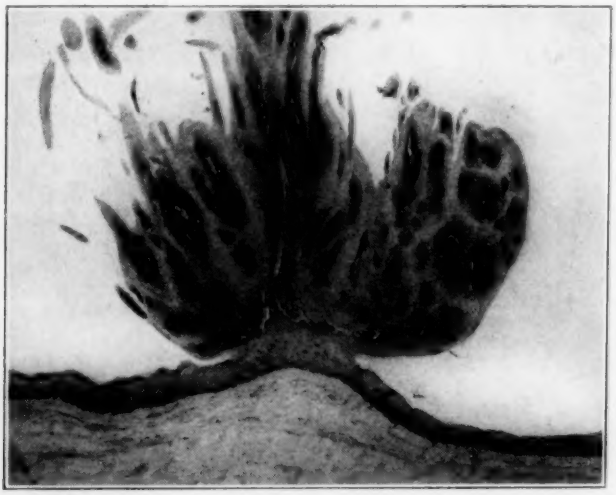

FIG. 2. Papilloma growing out from the mucous lining of the esophagus of a cow. Note the horny hair-like deposits on the surface (x 6). (Photomicrograph, courtesy of Dr. Wm. H. Feldman, Mayo Foundation, Rochester, Minn.)

REPORT OF A CASE

On postmortem inspection of a mature cow at a slaughtering establishment operating under federal inspection in Ogden, Utah, Dr. J. M. Allen, veterinary inspector, found the mucous membrane of the esophagus studded with numerous rough, hair-like tumors. The growths varied in size from that of a small pea to a fair-sized marble. The smaller tumors were pedunculated, while the larger ones were attached to the lining membrane by a rather broad base (fig. 1). A portion of the esophagus was forwarded to the Denver laboratory for diagnosis by Dr. F. G. Miller, inspector-in-charge at the Ogden station.

Sections prepared from several of the growths showed the usual microscopic picture of a papilloma, being composed of many

projections consisting of fibrous cores surrounded by several layers of epithelial cells (fig. 2). Excessive keratinization was seen in the sections, imparting a horny, hair-like appearance to the gross lesions.

REFERENCES

[1]Feldman, W. H.: Neoplasms of Domesticated Animals. W. A. Saunders Co., Philadelphia, (1932) p. 270.
[2]Lisi, G.: Papilloma of the urethra and bladder in a cow. Abst. Amer. Vet. Rev., xxxvii (1910), pp. 256-257.

SPECIAL TECHNICS FOR DUBBING AND CROPPING CHICKENS*

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The terms "dubbing" and "cropping" are used by poultrymen to indicate, respectively, the amputation of the comb and the wattles of a chicken. These appendages are removed, primarily,

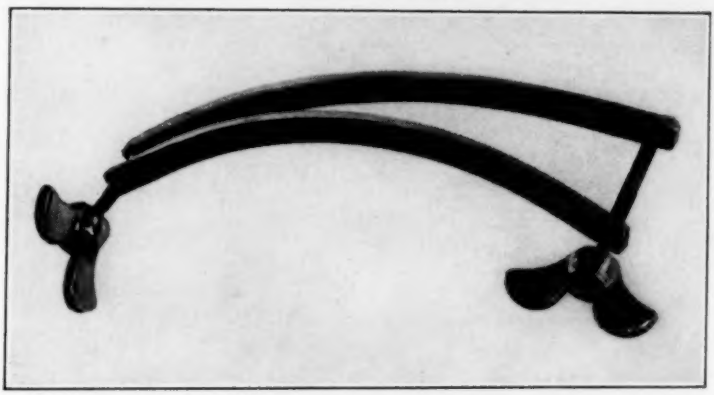

FIG. 1. Clamp.

to prevent the occurrence of frozen combs and edema of the wattles or for the treatment of these conditions after they have developed. Edema of the wattles is a frequent and often serious complication of infectious coryza caused by *Hemophilus gallinarum.*[1] It may result also from localized infection with *Pasteurella avicida*[2] or, probably, other species of bacteria. Because of the greater size of the comb and wattles in males, they are prone to be more susceptible to these conditions than females and, therefore, are more frequently subjected to dubbing and cropping.

*Received for publication, October 12, 1936.

Amputation of the comb is followed by profuse and sometimes fatal hemorrhage unless a proper technic is employed. The method to be described has been used on 63 full-grown males without a single death. It involves the use of a local anesthetic, a scalpel, a special clamp (fig. 1) and a searing-iron. The clamp consists of two curved steel bars and two bolts with winged heads. The bars are 11 cm long, 0.5 cm wide and 0.3 cm thick.

FIG. 2. Cockerel, with dubbing clamp in position.

They are curved inward to increase the pressure obtained at the center when they are in position and their inside surfaces are serrated to facilitate gripping of the tissues. The front bolt is 2 cm long and the back one 3.5 cm, exclusive of their heads. The holes in the bar adjacent to the boltheads are oblong to allow sufficient movement of the bolt in a horizontal plane, so that contact with the threaded holes in the opposite bar can readily be made when the clamp is applied to the comb. The

comb is anesthetized by hypodermic injection of a 2 per cent solution of novocain in combination with 0.005 per cent suprarenin.* The dose is varied between 2 and 3 cc, depending upon the size of the comb, and is injected in several places along its base. Surgical anesthesia develops within 10 minutes, after

FIG. 3. Cockerel, 24 hours after being dubbed.

which the clamp is applied and tightened sufficiently to stop the flow of blood into the appendage (fig. 2). The comb is then amputated by an incision one-eighth of an inch above and following the pattern of the clamp, and the cut surface thoroughly seared with a hot iron. The clamp is removed and the small

*Tablets containing 0.02 grams of novocain and 0.00005 grams of suprarenin were used. One tablet, dissolved in 1 cc of water, gave a solution containing 2 per cent novocain and 0.005 per cent suprarenin. The birds were injected with the anesthetic in groups of five. By the time the last bird in the group had been injected, sufficient time had elapsed so that the first bird injected was ready to be dubbed.

portion of comb remaining at the back is cut away and seared. Figure 3 shows a bird 24 hours after it has been dubbed. No ill effects were observed in any of the 63 birds which were dubbed by this method. The wounds, as a rule, were completely healed within 30 days.

The wattles can be removed easily and with safety with a pair of ordinary tin shears, followed by the application of an astringent, such as iron subsulfate (Monsel's solution). Occasionally a strong arterial hemorrhage is encountered but this may be controlled by applying a hemostatic forceps. The wattles can be removed at any age, but less hemorrhage is encountered if it is done before the bird reaches maturity.

REFERENCES

[1]Beach, J. R., and Schalm, O. W.: Studies of the clinical manifestations and transmissibility of infectious coryza of chickens. (In press.)

[2]Hughes, T. P., and Pritchett, I. W.: The epidemiology of fowl cholera. III. Portal of entry of *P. avicida;* reaction of host. Jour. Exp. Med., li (1930), p. 239.

PULLORUM DISEASE IN CAPTIVE QUAIL*

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An opportunity was recently presented for an investigation of losses which have been occurring throughout the 1936 growing season in young quail on a farm maintaining about 300 pairs of breeding birds. During the 1935 rearing season, when eggs from wild stock were hatched, very few losses occurred. During the 1936 season, however, considerable difficulty was encountered. In March, 1936, 100 pairs of outside stock raised in captivity were purchased for breeding purposes.

Eggs used for hatching in the spring of 1936 were fertile but had poor hatchability; many embryos developed but failed to emerge completely from the shell, while others died in the shell. There was an increase in the number of crippled and weak chicks. An unusually high mortality occurred during the first ten days of life. The course of the disease after the development of symptoms was rapid in most instances; some chicks recovered but many presented an unthrifty appearance. Better livability was secured from chicks hatched in "still air" incubators than in an incubator through which air was circulated by a fan.

Five affected chicks autopsied showed unabsorbed egg yolks. Three chicks showed small pin-point brown foci on the surface

*Received for publication, November 5, 1936.

of the liver. Two chicks showed a few small irregular brown foci on the lungs. Hemocytoblastosis was observed in blood-smears from all the chicks. The affected birds showed unthriftiness, drooping wings and unsteady gait; one showed wry neck. Cultures from the organs of two of these chicks yielded a micro-organism showing characteristics of *Salmonella pullorum.*

Whole-blood stained pullorum antigen tests were made on ten pairs of birds secured in March, 1936; three reactors were found. Similar tests were made on ten pairs of birds reared during the 1935 season from wild stock; two reactors were found. Two reactors were also found in ten females, 21 weeks of age, reared during the present season. The reactions shown in these tests were not so distinct as usually observed in chickens. There may be a number of factors involved. The birds were in a non-producing period when tested; the causal microörganism was in an unusual environment; infected quail may not have marked ability to produce agglutinins.

It seems that the infection was apparently introduced to the premises of this farm in the birds received in March, 1936, and that the infection was spread to a considerable extent by means of the incubators. Information is at hand that the farm from which the breeding birds were purchased in March, 1936, experienced difficulties somewhat similar to those described above but no knowledge is at hand as to the actual cause.

Giltner[1] reports that Morley found the quail relatively resistant to artificial *S. pullorum* infection but that in a few instances he was able to induce infection.

While it is not likely that pullorum disease is of particular importance in wild quail at this time, the release of pullorum-infected birds from quail farms is a potential menace to the future welfare of this game bird, particularly at this time, when the restocking of the wild supply is a conservation program of many states.

REFERENCE

[1]Giltner, L. T.: Personal communication.

Specialist or Scientist

A Specialist is one who knows more and more about less and less until he knows everything about nothing. A Scientist is one who knows less and less about more and more until finally he knows nothing about everything.

—*The World Call*

THE EFFECT OF MUCIN ON INFECTION BY BACTERIA. W. J. Nungester, L. F. Jourdonais and A. A. Wolf. Jour. Inf. Dis., lix (1936), p. 11.

When sterilized gastric mucin was used instead of saline as a menstruum for suspending various bacteria, the host was placed at a marked disadvantage and succumbed to what would otherwise be a sublethal infection. This effect was noted when injections were made intraperitoneally, subcutaneously or intratracheally. The mechanism of the action is not completely understood. Mucin does not interfere with phagocytosis but does inhibit the bactericidal properties of phagocytic cells. It enables bacteria to survive in the body of the host for longer periods without diminution in numbers or it may allow the organisms to increase in numbers and so result in the death of the animal. The viscosity and cohesive properties of the mucin appear to be important indices as to the effectiveness of mucin on bacterial infections.

PRECIPITIN AND COMPLEMENT-FIXATION REACTIONS OF POLYSACCHARIDE EXTRACTS OF BRUCELLA. Margaret Higginbotham and Lucy S. Heathman. Jour. Inf. Dis., lix (1936), p. 30.

The results of the precipitin tests with the polysaccharide preparations from seven strains of *Brucella* seem to show that organisms otherwise classified as to type may possess variable antigenic properties. The precipitin test is not a satisfactory one for establishing the type identity of a *Brucella* organism, although extracts from three of seven strains were found to give a positive reaction with the homologous type antiserum only. The results support the contention that a polyvalent antigen, including some local strains, should be employed in the routine serological examination for undulant fever. All the serums from cases of human brucellosis which showed agglutination with the stock antigens *(Br. melitensis, abortus* and *suis)* gave positive precipitin reactions with one or more of the *Brucella* polysaccharides. Although the series was small, the polysaccharide precipitin test would seem

to have no advantage over the agglutination test as a routine procedure and has the disadvantage of being impractical because of the time and cost involved in preparing extracts. The results of the complement-fixation tests with the *Brucella* polysaccharide extracts and specific antiserums, as well as those with the extracts and patient's serum, were not so clear cut as the precipitin reactions.

THE BACTERICIDAL AND BIOLOGICAL PROPERTIES OF PHENYL MERCURIC SUBNITRATE. K. T. Sasano and E. M. Medlar. Jour. Inf. Dis., lix (1936), p. 35.

Phenyl mercuric subnitrate, aside from being a powerful germicide, has the desirable quality of non-interference with the antigenic properties or immunological substances. It is relatively non-toxic. However, a 10-cc dose in a 1:1,500 solution of phenyl mercuric subnitrate causes a severe local inflammation at the site of intravenous injection but there is no apparent systemic reaction.

STUDIES ON TETANUS TOXOID. III. Antitoxic response in guinea pigs immunized with tetanus alum-precipitated toxoid followed by tetanus spores. F. G. Jones and A. Jamieson. Jour. Bact., xxxii (1936), p. 33.

Guinea pigs that have received one or more injections of tetanus alum-precipitated toxoid and are infected within two months are protected against a massive dose of tetanus spores which kills normal pigs in from 88 to 112 hours. A massive dose of tetanus spores does not markedly accelerate the production of antitoxin in guinea pigs that have been previously immunized with tetanus alum-precipitated toxoid.

THE RECOVERY OF VIRULENT TUBERCLE BACILLI FROM THE TISSUES OF SWINE INTENDED FOR FOOD. William H. Feldman. Jour. Inf. Dis., lix (1936), p. 43.

Attempts were made to isolate and type tubercle bacilli that might be present in dressed carcasses of swine that had been retained on postmortem inspection for tuberculosis. Material was obtained from only those carcasses in which the lesions of tuberculosis were slight or localized and which were "passed for food" after the diseased tissues had been removed. The left precural lymph-node from each of 88 carcasses and the right

internal iliac lymph-node from each of 13 carcasses were obtained and cultures prepared. Of the total of 111 specimens, tubercle bacilli were obtained from four. Studies on pathogenicity revealed all of the strains to be *Mycobacterium tuberculosis* avium. The author suggests the importance of the problem of avian tuberculosis and the indications that virulent tubercle bacilli may still be present in the carcasses of hogs even though all visible lesions of tuberculosis have been removed.

STUDIES OF STREPTOCOCCI. IV. Resistance of enterococci. George H. Chapman. Jour. Bact., xxxii (1936), p. 41.

The enterococcus (syn. *Streptococcus fecalis, Micrococcus ovalis* and possibly *Streptococcus lactis*) was resistant to a number of injurious agents. Of 75 strains tested, 70 resisted the action of 1:500,000 merthiolate, 1:2,500 phenol, 1:5,000 basic fuchsin, 1:50,000 hexylresorcinol and 1.0 per cent sodium carbonate for one hour. In contrast, only one out of 16 smooth streptococci was resistant to these agents. When compared with *Escherichia coli, Aerobacter aerogenes,* staphylococci and streptococci, enterococci were the most resistant to sodium carbonate, sodium bicarbonate and sodium chloride. Enterococci were still viable after contact for one hour with 3.0 per cent sodium carbonate. Enterococci usually did not kill rabbits when 5 cc of their live brain-heart infusion cultures were injected intravenously. Smooth streptococci that were resistant to injurious agents usually killed rabbits under similar conditions. The interpretation of resistance to injurious agents would be simplified if enterococci are differentiated from other streptococci.

STUDIES ON THE NATURE OF IMMUNITY TO INTESTINAL HELMINTHS. IV. The inter-relations between parenteral and intestinal immunity in rats infected with *Nippostrongylus.* Asa C. Chandler. Amer. Jour. Hyg., xxiv (1936), p. 129.

Parenteral vaccines have a marked effect not only on the number of worms which reach the intestine after subsequent inoculations but also on the growth and reproduction of the worms after their arrival in the intestine. Vaccines are more effective when prepared from worms killed in saturated salt solution than when hydrogen peroxide is used to kill them. The ingestion of dead adult and larval worms over a period of five days had no appreciable effect on the course of a subsequent infection. The

gradual disintegration of *Nippostrongylus muris* transplanted into the body cavity stimulates a small degree of immunity, reducing the number of worms which reach the intestine after subsequent inoculations and lowering their egg production. In rats with normal intestinal infections developing after the usual parenteral migration phase, worms transplanted into the body cavity are disintegrated more rapidly than in previously uninfected rats.

This accelerated reaction of the body tissues is probably due to stimulation resulting from destruction of larvae during the parenteral migration phase, since no accelerated disintegration of body cavity worms was observed in rats which had had worms transplanted directly into the intestines. Rats which have had worms transplanted directly into the intestines show a marked reaction against subsequent infections not only by a reduction in egg output but also by a retardation of growth and development, though the effect is not so marked as that following a normal subcutaneous infection. While a local intestinal immunity may develop which is not shared by the blood, and so does not become general, a parenterally developed immunity is communicated to the intestinal wall and affects the growth and reproduction of parasites situated in the intestine.

CONCERNING THE TRANSMISSION OF THE FIBROMA VIRUS (SHOPE) OF RABBITS. Roscoe R. Hyde. Amer. Jour. Hyg., xxiv (1936), p. 217.

The domestic rabbit, in which the fibroma virus has been established in the upper respiratory tract, fails to transmit this disease by contact as shown by susceptibility of the contact rabbits to the myxoma virus. This also holds true when the fibroma virus is established by way of the skin or the eye. The cottontail treated with the myxoma virus by way of the eye, the skin or the nares, fails to transmit this disease to the domestic rabbit by contact. The myxoma virus was apparently effective in the cottontail as shown by tests with the fibroma virus. The domestic rabbit treated with the fibroma virus by way of the eye, nose or skin, is highly immune to the myxoma virus by all portals of entry. Fibroma-immune rabbits, which may show marked myxoma symptoms and recover, free themselves of the myxoma virus for their tissues after a time are ineffective in transmitting the disease. Heated myxoma virus was not activated by the addition of fresh fibroma virus.

STAPHYLOCOCCAL IMMUNITY. J. S. Kitching and L. N. Farrell. Amer. Jour. Hyg., xxiv (1936), p. 268.

Active immunization of rabbits and mice with staphylococcus toxoid produced increased resistance against the lethal effect of staphylococci introduced parenterally and also against the dermo-necrotic effect of the staphylotoxin introduced intradermally in rabbits. In general the protection against these two effects depended upon the amount of circulating antitoxin as determined by serum titrations using the standard unit as a basis for comparison. The titre of circulating antitoxin fell off approximately ten days after the last of a course of injections of toxoid in rabbits. The drop was at first sharp, to approximately half, and then became gradual. The titre could be raised approximately to its former maximum by one further injection of toxoid.

Injection of rabbits and mice with staphylococcus formalinized vaccine, prepared and used under the conditions described, produced little or no circulating antitoxin and no increased resistance against the lethal effect of staphylococci and staphylotoxin introduced parenterally or against the dermo-necrotic effect of staphylotoxin in rabbits. The serum obtained after the injection of rabbits with staphylococcus formalinized vaccine in the quantity used did not confer passive immunity against the lethal effect of staphylococci in rabbits and mice. Staphylococcus antitoxin was found to be a definitely specific agent for conferring passive immunity against the lethal effect of living staphylococci and staphylotoxin introduced parenterally into rabbits and mice. The immunity was in direct proportion to the number of units of antitoxin given. Antitoxin given intravenously before the injection of toxin was found to have a modifying effect upon damage to tissues by the toxin and under the conditions of the experiment, definitely to prevent the lethal effect of toxin or microörganisms. Delay in administration of antitoxin, particularly where the subcutaneous route was used, distinctly lessened its value.

STUDIES ON THE BACTERICIDAL ACTION OF PHENOL AND MERTHIOLATE USED ALONE AND IN MIXTURE. Carolyn R. Falk and Sophronia P. Aplington. Amer. Jour. Hyg., xxiv (1936), p. 285.

The effect of using phenol and merthiolate in mixture is additive. Mixtures of selected concentration of phenol and merthiolate will act on all three test organisms, diphtheroids, staphylococci and *B. pyocyaneus*. From the data presented it is possible

to select the relative concentration of phenol and merthiolate required to kill the indicated organisms under definite conditions. The general phenomena of disinfection, in connection with the influence of time, temperature and medium on bactericidal action, may be applied to the mixtures of the antiseptics. The necessity of using a solid as well as a liquid medium to test the presence of living organisms is again demonstrated, although no evidence is presented to form any definite conclusions as to the causes for this observation.

EFFECT OF MINERALS AND FIBER ON AVIAN INTESTINAL pH. V. G. Heller and Robert Penquite. Poultry Sci., xv (1936), p. 397.

The digestive tract of the fowl is equipped to care for all normal changes in the food and water ingested. Large amounts of basic salts in the feed or water do decrease the acidity of the crop, proventriculus and gizzard, but the changes are of small significance in the intestine. Changes in the calcium-phosphorus ration, such as would ordinarily be produced in the metabolism of varying amounts of protein and fiber, must be attributed to factors other than the pH of the digestive tract.

THE INCIDENCE OF BLINDNESS AND PARALYSIS ACCORDING TO FAMILY. Chas. W. Upp and B. A. Tower. Poultry Sci., xv (1936), p. 421.

Although great variation was noted in the incidence of blindness and paralysis in 528 mature progeny from 60 dams mated to seven sires in 1934, there were apparently no family differences. The distributions were abnormal enough to indicate inherent differences in resistance to disease. The incidence of blindness considered on a family basis was studied in the case of 60 dams and nine sires. The sires used produced results more variable than can be attributed to chance alone. In two cases of twelve, the progeny of dams mated to the same male differed significantly in the incidence of the disease. The incidence of blindness was as follows: from parents with affected eyes, 70 per cent affected; from flock-run parents, 67.9 per cent affected; from questionably susceptible family parents, 54.6 per cent affected; from questionably resistant parents, 47.4 per cent affected. The contention that blindness is merely a late manifestation of paralysis is not tenable for the data obtained in these experiments. Blindness was much more prevalent than paralysis in the matings and some families that had a high incidence of blindness showed paralysis.

Regular Army

Major Forest L. Holycross is relieved from further assignment and duty at Fort Hayes, Ohio, and from additional duty at headquarters, 5th Corps Area, and the General Reserve Depot, Columbus, Ohio, effective on or about January 1, 1937, is then assigned to station at Fort McClellan, Ala., and will proceed to Washington, D. C., and report to the commanding general, Army Medical Center, for temporary duty for a period of approximately one month for the purpose of pursuing a post-graduate course of instruction at the Army Veterinary School; upon completion of this duty will proceed to Fort McClellan, Ala., and report to the commanding officer for duty. Major Holycross is, in addition to his duties at Fort McClellan, Ala., detailed as attending veterinarian, Maxwell Field, Ala.

Colonel Alfred L. Mason is relieved from further assignment and duty as station veterinarian, Presidio of San Francisco, Calif., and from additional duty at headquarters, 9th Corps Area, and will report to the commanding general 9th Corps Area, Presidio of San Francisco, Calif., for duty with the Veterinary Corps at his headquarters, and additional duty as station veterinarian, Presidio of San Francisco.

Veterinary Reserve Corps

New Acceptances

Mitchel, Charles A.....1st Lt...Cheyenne, Wyo.
Winsor, Frank Reed.....1st Lt...9 Prospect St., Montgomery, Ala.

Promotions

To

Swanson, Leonard E.....Capt...Univ. of Hawaii Exp. Sta., Honolulu, T. H.
Brenner, Carl F.....1st Lt...1210 Macklind, Saint Louis, Mo.
Karlson, Alfred G.....1st Lt...227 Sheldon, Ames, Iowa.
Mohr, Earl S.....1st Lt...R. 3, Connersville, Ind.

New Assignments to Active Duty with CCC

Fly, Glen O.....1st Lt...Fort Knox Dist., Fort Knox, Ky.
Currie, Arthur J.....Capt...Chicago QMD, Chicago, Ill.
Schwiesow, Carlton W.....1st Lt...Chicago QMD, Chicago, Ill.
Talcott, Robert V.....1st Lt...Pres. of San Francisco, Calif.

Termination of Assignment to Active Duty

Sample, Fred M.....1st Lt...Tyler Dist., Tyler, Texas.
Orr, Norton A.....1st Lt...Fort Douglas, Utah.

SECURITY FROM TUBERCULOSIS

By PHILIP P. JACOBS

Your chances of dying from tuberculosis were about four times as great some 30 years ago as they are today. Thus you have today gained a security from tuberculosis such as the world has never before known.

Thirty years ago, 200 out of every 100,000 people living in the United States were dying every year from tuberculosis. If you go back another 30 years the number is more than 300, or three out of every 1,000 people who lived and worked in the days of your mothers and fathers! No wonder Oliver Wendell Holmes called it "The White Plague"! Today, not many more than 50 people are dying from this disease yearly in each 100,000. Still a large number, to be sure, but small by way of comparison with figures of a generation or two generations ago.

Thirty years ago, the estimated number of living persons in the United States who had tuberculosis was approximately 1,500,000. Today, there is less than one-half that number of living victims of this disease in this country. When it is realized that every victim of tuberculosis is a potential spreader of tuberculosis to an average of three others, the chances of "catching" the disease today, as compared with 30 years ago, are very much less. Here is increased security indeed.

You, Mr. Taxpayer, and you, Mr. Citizen, have helped the National, state and local tuberculosis associations to buy this security from tuberculosis for your state and community. For through your support, hospitals and other institutions have been built in every state of the Union, some 1,200 in all, with 95,000 beds for the care of tuberculosis victims. You have by your taxes helped to build these institutions and you are helping to maintain them. And there are today more than 1,000 clinics,

and 10,000 public health nurses, besides a great army of doctors, social workers, and other persons and agencies, all of whom are working with you and for you to help to keep tuberculosis away from your family and you.

But this happy result has not been brought about by accident. Thirty years ago, the first tuberculosis Christmas Seal in the United States was sold in Wilmington, Delaware. That little holiday sticker made possible the formation of hundreds of tuberculosis associations (today, 1,981) and these groups of citizens working patiently day after day have made you and millions like you realize that health can be bought. The proceeds from the Christmas Seal sales for 30 years would be relatively insignificant, when compared with a year's expenditures by the federal government, for example. And yet they have helped to give you and yours a security from tuberculosis that is of inestimable value.

Tuberculosis still is one of our greatest, and in many communities our greatest, health hazard. It still takes a toll of almost 70,000 lives annually. It still numbers its living victims by more than 500,000, and it still causes loss of life and health that costs our country at least three-quarters of a billion dollars a year. We have enough scientific knowledge to bring this "White Plague" under control, if not to eradicate it. To do this requires the education of many millions of people, in order to get them to do their part in ridding the country of tuberculosis. For this purpose the tuberculosis associations of the United States, local, state and National, appeal to you to buy Christmas Seals this year.

Classmates

One came from his school with polish and poise
And counted his work as his greatest of joys,
Aseptic, precise, and exact to a minim
But the other guffawed and swore that to "win 'em"
One must be a "mixer" and easy to meet;
Never mind if a little dung clung to one's feet.

He'd jab in a needle regardless of rust
And would say of an abscess, "Ah! just let her bust."
They both made a living, these two college boys,
The one ostentatious, the other with poise,
Would dole out their pills, aseptic or not,
And each from his practice got just what he got.

M. L. PLUMER

EAST CENTRAL (IOWA) VETERINARY ASSOCIATION

At the meeting of the East Central Veterinary Association held at the Hotel Montrose, Cedar Rapids, Iowa, September 10, 1936, 47 veterinary practitioners were present from 15 counties. The program was a symposium on various forms of poisoning among live stock.

Dr. F. M. Wilson, of Mechanicsville, reported cases of forage poisoning in sheep, cattle and horses. He also covered poisoning by moldy ensilage and black cherry leaf hedge trimmings. He also reported on cottonseed meal poisoning in steers. Dr. I. L. Hawkins, of Cascade, reported on cornstalk poisoning and cockle burr poisoning in cattle. He showed specimens on whorled milkweed and cockle burr shoots.

Dr. J. S. Potter, of Iowa City, gave some case reports on lead poisoning in cattle and swine and moldy hay poisoning in steers. Dr. Edw. H. Beretta, of Solon, reported on Sudan grass poisoning in cattle.

Dr. A. H. Quin, Jr., of Des Moines, talked on poisoning by cane, millet, cornstalks and oak leaves, in horses and other animals. Dr. C. B. Strain, of Dunkerton, rendered a case report on forage poisoning in horses. Dr. C. H. Banks, of Tipton, covered cherry leaf poisoning in cattle.

Dr. R. M. Hofferd, of Cedar Rapids, reviewed belladonna plant (deadly nightshade) poisoning in pigs. Dr. G. A. Kay, of Cedar Rapids, reported cases of poisoning in cattle caused by eating ensilage contaminated by paint from the inside of a silo. He also gave case reports on burdock poisoning and presented some cases of poisoning caused by canning factory refuse.

Dr. F. F. Meads, of Oskaloosa, covered jack oak poisoning in horses and mules. Dr. W. E. Stribling, of Des Moines, gave the postmortem lesions found in cases of poisoning by prussic acid. Dr. John B. Bryant, of Mount Vernon, gave a report of a case of poisoning by cornstalks that had been cut and allowed to lie before being fed to cattle. Dr. N. A. Kippen, of Independence, reported on salt poisoning and cockle burr poisoning in swine, and acorn poisoning in cattle.

Dr. A. R. Menary, of Cedar Rapids, gave the results of testing 300 herds of cattle in Linn county for Bang's disease. With the first test, 1,284 of the 6,122 animals in these herds reacted, a percentage of 20.9. At the second test, 9.6 per cent reacted and only 7.8 per cent with the third test (second retest).

Dr. H. A. Seidell, chief of the Bureau of Animal Industry, Iowa Department of Agriculture, addressed the meeting on "The Regulation of Community Live Stock Sales." Dr. W. S. O'Brien, of Ryan, president of the Eastern Iowa Veterinary Association, discussed plans for the technical program for the October meeting of that organization.

The election of officers resulted as follows: President, Dr. Samuel G. Paul, Clarence; vice-president, Dr. L. Proctor, Hazleton, and secretary-treasurer, Dr. Edw. H. Beretta, Solon.

SOUTHWESTERN MINNESOTA VETERINARY MEDICAL ASSOCIATION

The regular semi-annual meeting of the Southwestern Minnesota Veterinary Medical Association was held in the City Hall, Windom, Minn., September 24, 1936, with about 60 veterinarians in attendance. Dr. D. T. Grady, of Windom, president of the Association, presided.

The following papers were presented:

"Immunity, Especially as It Pertains to Hog Cholera," by Dr. H. C. H. Kernkamp, University of Minnesota, Saint Paul.

"Some New Trends in Therapeutics," by Dr. L. A. Merillat, Chicago, Ill.

"Ways and Means of Increasing Revenue in Country Practice," by Dr. A. H. Quin, Fort Dodge, Iowa.

Eight new members were admitted to the Association. An amendment to the by-laws was adopted providing for a registration fee of one dollar being charged non-members who attend meetings of the Association.

A banquet for the veterinarians and their wives was served in the Park Hotel following the meeting. Dr. Kernkamp very ably presided as toastmaster.

L. E. STANTON, *Secretary.*

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The October meeting of the Veterinary Medical Association of New York City was held at the Hotel New Yorker, Wednesday evening, October 7, 1936.

We were fortunate in having the pleasure of hearing two distinguished and outstanding speakers, each with a very interesting subject. The first was Dr. R. L. High, president of the High Chemical Company, Philadelphia, Pa., who presented a paper entitled "Solution Normet, an Artificial Serum Based on Citrates Which Permits the Final Survival of Bled Animals." Dr. High discussed the chemistry and physiology of and the uses and indications for Solution Normet. The paper for practical purposes can best be summed up by applying the slogan of the manufacturer of Solution Normet, which states that "when you think of blood transfusions, use Solution Normet." This is the purpose of the product and can be looked to for results in postoperative shock, hemorrhages and similar conditions.

The second speaker of the evening was Dr. Harry W. Jakeman, of Boston, Mass., who came to us with the timely topic, "Some Phases of Rabies and Distemper." Dr. Jakeman discussed the problems of both diseases from the standpoint of what the veterinarian could and should do in controlling diseases and problems which are wholly his. An instance is the proper procedure for promoting vaccination against rabies. Another is some intelligent and sensible manner in which the public can be informed properly of the benefits to be derived from such measures. Should every veterinary organization concentrate on educating its community, as has been done by the Massachusetts Veterinary Association, the "rabble rousers" in questions of public health would quickly be quashed. As correctly stated by Dr. Jakeman, the solution of the question lies with veterinarians themselves. It is their problem to inform dog-owners properly.

The subject matter expressed by both speakers was heartily received and they were tendered a rising vote of thanks for their splendid presentations.

R. S. MacKellar, Jr., *Secretary.*

NEW ENGLAND VETERINARY MEDICAL ASSOCIATION

The seventh annual convention of the New England Veterinary Medical Association was held in Providence, R. I., October 14-15th, 1936. A full and interesting program was provided, covering many phases of veterinary practice. About 120 members were present.

At the clinic, Dr. W. J. R. Fowler, of Guelph, Ontario, operated on a number of horse cases; Dr. C. P. Zepp, of New York

City, discussed a number of small-animal cases and gave several demonstrations, while Dr. E. F. Schroeder, of Boston, Mass., demonstrated the use of the Schroeder splint in replacement of several dislocated hips, as well as a fracture of the femur.

Dr. J. P. Delaplane, of the Rhode Island State College, gave a very interesting demonstration of poultry diseases and had on hand a display of pathological specimens. Dr. J. D. Jones, of Providence, R. I., demonstrated a number of pathological specimens obtained from small-animal practice. It was voted by those in attendance as the most interesting and instructive clinic which the Association has ever held.

At the business session of the Association, it was voted to offer a medal for a child under 15, residing in New England, who has performed the most humane act in connection with animals during the year. This will be given publicity in the schools throughout the New England states. The nominations will be made by humane societies in the different states and final judgment made by the Executive Board of the New England Veterinary Medical Association. It is proposed to give the matter considerable newspaper publicity, to pay the way of the child to the meeting, and have the presentation made by some prominent state official.

An interesting occurrence during the meeting was the presentation of an equipped traveling bag to Dr. W. J. R. Fowler as an expression of the Association's appreciation of his work in connection with several of the annual meetings. This presentation was made for the Association by Dr. P. R. Baird, of Waterville, Me., and the members were interested in learning that Dr. Fowler is the only English-speaking veterinarian who has had conferred upon him the honor of Chevalier du Mérite Agricole de France by the government of France.* This honor was bestowed upon Dr. Fowler last year.

The following officers were elected for the ensuing year: President, Dr. L. A. Paquin, Webster, Mass.; secretary-treasurer, Dr. H. W. Jakeman (reëlected), Boston, Mass. The vice-presidents who make up the Executive Board, are the presidents of the six New England state associations: Dr. R. E. Libby, Richmond, Me.; Dr. F. V. Dederick, Keene, N. H.; Dr. J. G. Richardson, Providence, R. I.; Dr. F. J. Brockett, Suffield, Conn.; Dr. H. L. Mills, Burlington, Vt., and Dr. F. A. Miller, Fitchburg, Mass.

H. W. JAKEMAN, *Secretary.*

*Jour. A.V.M.A., lxxxvii (1935), n. s. 40 (5), p. 612.

PURDUE UNIVERSITY VETERINARY SHORT COURSE

A short course for veterinarians was held at Purdue University, West Lafayette, Ind., October 20-23, 1936. The program consisted of laboratory demonstrations, discussions of animal diseases and related subjects, and a clinic. About 150 veterinarians attended the short course.

The first session began at 1:30, Tuesday afternoon. All the afternoon was taken up by a laboratory in hog diseases. Members of the veterinary staff, assisted by Dr. V. F. Saylor, of the State Live Stock Sanitary Board, autopsied the hogs and led in the discussion. Diseases of cattle were discussed the second and third days of the course. A part of Wednesday afternoon was taken up by a laboratory in judging dairy cattle. Prof. E. T. Wallace, of the Dairy Department, was in charge of the judging work.

The following discussions should be given special mention: "Diseases of the Digestive Tract of Cattle," by Dr. J. L. Axby, State Veterinarian; "Avitaminosis," by Dr. S. M. Hauge, of the Purdue University research staff; "Dairy and Milk Inspection," by Mr. John Taylor, Chief of Bureau of Dairy Products, State Division of Public Health; and "Bang's Disease," by Dr. H. Busman, U. S. Bureau of Animal Industry. The dinner meeting was held in the Purdue Memorial Union Building. Following the dinner, Dr. E. E. VanLone, of the School of Agriculture, addressed the veterinarians on the subject of "Hereditary Lethals."

In his address of welcome, Dean J. H. Skinner mentioned the work of the different departments in the Agricultural Experiment Station, and emphasized the need for more research work that is fundamental or basic. The address by Dr. H. Preston Hoskins, secretary-editor of the American Veterinary Medical Association, was of special interest. In discussing the large increase in the enrollment in veterinary colleges, he stated that the veterinary profession is now generally considered to be the least crowded of the professions, and that high school graduates are given this information by their advisors and vocational directors.

Dr. W. B. Craig, of Indianapolis, had charge of the clinic. Because of the interest in the clinic, more emphasis will be given this part of the program in planning future short courses.

R. A. CRAIG, *Reporter.*

INTERSTATE VETERINARY MEDICAL ASSOCIATION

The Interstate Veterinary Medical Association broke all existing records at its meeting in Sioux City, Iowa, October 22-23, 1936, when 184 veterinarians registered and 175 persons were at the banquet. About 60 per cent of the veterinarians in attendance were from Iowa and the balance were chiefly from Nebraska, South Dakota and Minnesota.

Dr. E. L. Eggleston, of Alcester, S. Dak., presided and kept the program going on schedule, which seemed to be very much appreciated. Among those who participated in the program on the first day were: Dr. H. Preston Hoskins, secretary-editor of the American Veterinary Medical Association, who discussed several matters in connection with veterinary education, veterinary student enrollment, publicity for veterinarians, and a plan for reporting animal diseases inaugurated in Illinois; Dr. E. R. Frank, of Kansas State College, who gave an illustrated lecture on "Lamenesses in the Horse"; Dr. A. T. Kinsley, of Kansas City, who discussed "Post-Vaccination Troubles in Swine," and Mr. Harry Linn, field man for the Iowa Horse and Mule Breeders' Association. Mr. Linn related his experiences on a recent trip to Europe to purchase a herd of Suffolk Punch horses. He illustrated his talk with a motion-picture film, most of which was taken in England. This was one of the high spots of the meeting.

On the second day, Dr. J. E. Weinman, of Lincoln, Neb., led off with one of his interesting discussions of problems met with in small-animal practice. Dr. P. V. Neuzil, of Blairstown, Iowa, told how he had built up a large poultry practice by coöperating with local hatcheries. Dr. H. D. Bergman, of Iowa State College, discussed practical veterinary therapeutics in a novel manner. He presented each veterinarian with a mimeographed set of notes on pharmacology and then pointed out important features in modern use. At the afternoon session, Dr. J. B. Taylor, of South Dakota State College, demonstrated the pregnancy test upon rabbits. He then used lantern-slides to illustrate methods of procuring and examining semen of the bull. Dr. I. J. Kleaveland, of Cedar Rapids, Iowa, discussed many of the common problems and some unusual cases met with in cattle. Dr. L. B. Frederick, of Swift and Company, Chicago, gave a talk on sheep.

At the banquet Thursday evening, Dr. H. D. Bergman presided as toastmaster and Dr. W. T. Spencer, of Lincoln, Neb., chairman of the Committee on Local Arrangements for the 1937 meeting of the A. V. M. A. in Omaha, was the principal speaker.

Dr. T. W. Munce, of Sioux City, Iowa, was elected to represent the Association on the Committee on Local Arrangements.

Officers elected for the coming year were as follows: President, Dr. C. H. Haggard, of Luverne, Minn.; vice-president, Dr. E. R. Truax, of Sac City, Iowa, and secretary-treasurer, Dr. W. A. Aitken (reëlected), of Merrill, Iowa.

The Ladies Auxiliary elected officers as follows: President, Mrs. G. B. Fincham, of Sioux City, Iowa; vice-president, Mrs. A. A. Fosterman, of Utica, S. Dak.; secretary, Mrs. S. S. Gibson, of Randolph, Neb., and corresponding secretary, Mrs. M. F. Wallace, of Sioux City, Iowa.

W. A. AITKEN, *Secretary.*

MICHIGAN-OHIO VETERINARY MEDICAL ASSOCIATION

The semi-annual meeting of the Michigan-Ohio Veterinary Medical Association was held in Blissfield, Mich., on October 29, 1936. There were 32 veterinarians in attendance, 14 from Ohio and 17 from Michigan.

The program included a very excellent discussion of poultry practice and an outline of the various compounds which may be used in such a practice. This was given by Dr. H. E. Ash, of Bowling Green, Ohio. In his talk, he gave some very good formulas which may be used by practitioners who give much time to poultry work.

The next subject on the program was a paper by Dr. N. D. Backus, of Elyria, Ohio. His subject was "Lameness with Especial Reference to Acute Laminitis." Dr. Backus spent considerable time on this subject and the paper deserves commendation for its excellence. He brought out in his discussion the differential diagnosis between laminitis and myositis. (This paper will be published in the JOURNAL.)

Dr. C. W. Witty, of Elmore, Ohio, gave a blackboard illustration of his method of castrating cryptorchid pigs. It is planned to have several such pigs at the spring clinic of this Association so that Dr. Witty may demonstrate his skill.

Dr. S. G. Colby, of Monroe, Mich., is working on a program whereby the practicing veterinarian may be recognized in his work on Bang's disease testing. He gave to the Association the results of his endeavors.

Dr. Wm. G. Hansen, of Greenville, Mich., president of the Michigan State Veterinary Medical Association, attended this meeting and gave a talk about the cattle reduction program in

Michigan and how it affected the dairy products in this state by reducing the sale of butter and increasing the consumption of the substitutes.

The meeting was considered to have been highly instructive and was much appreciated.

E. C. W. SCHUBEL, *Secretary.*

Michigan Short Course

The fourteenth annual Postgraduate Short Course for Veterinarians at Michigan State College will be held during the week of January 25-29, 1937. According to an announcement by Dean Giltner, the program will include symposia on the rapid agglutination test for Brucellosis, vaccination against Brucellosis, and the rapid plate whole-blood agglutination test for pullorum disease.

It is planned to present what is known about the manufacture of the antigens used in the agglutination tests, and the most effective use of the antigens in the testing of blood samples. There will be a review of what is known about vaccination to control Brucellosis in cattle and other animals.

A cordial invitation is extended to veterinarians in all branches of the profession—practitioners, research workers, regulatory officials, commercial men—to attend and avail themselves of this wonderful opportunity to extend their knowledge and contribute to the discussions as well.

The program will not be confined to the subjects mentioned although the various phases of Brucellosis will be emphasized.

Good-Bye T. B. Cow

Soon you'll not see a T. B. cow,
That coughing, low-down critter;
The Vets now have her on the run,
Her old-time friends have quit her.
For spreading germs she is renowned,
Her milk it is polluted,
She isn't worth her board and keep,
For breeding she's not suited.
From states now numbering forty-two,
Crusaders, wise and clever,
Have banished her and sealed her doom
In U. S. A. forever.

RUDOLPH SPIRES ALLEN

NECROLOGY

GEORGE HOWARD DAVISON

Dr. G. Howard Davison, formerly of Millbrook, N. Y., died in Madison Newton Dorset, England, September 2, 1936, at the age of 69 years, after an illness of about a year. He had been living in England for about five years.

Dr. Davison was a graduate of the American Veterinary College, class of 1890, and was a member of the A. V. M. A. from 1893 to 1897. His portrait hangs in the gallery of the Saddle and Sirloin Club, of Chicago, and the following sketch of his career has been supplied by Mr. Edward N. Wentworth, director of Armour's Live Stock Bureau, Armour and Company, Chicago, under the title, "An American Who Invaded Shropshire."

Probably the pioneer Shropshire breeder of the United States from a constructive standpoint was Dr. G. Howard Davison. His foundations for the Altamont flock at Millbrook, N. Y., were laid in the best blood to be secured in Britain during the 90's, his purchases being from the Tanner, Bowen-Jones and Minton flocks. American breeders of those days leaned very strongly toward a big-framed, more slowly maturing, open-fleeced kind, but Dr. Davison from the start advocated a compact, thickly meated, early-finishing type that would fit more strongly into the niceties of consumer demand. His first sheep were brought over in 1893, under the care of his first shepherd, Herbert Fox. Ambitious to equal the productions of the Shrewsbury district, he sent to England the second year thereafter a flock of his own breeding for exhibition and competition at the English Royal.

So successful did this mission prove that Dr. Davison was paid high tribute by the British agricultural journals of the day and was elected a member of the Royal Agricultural Society. In the same year (1895), he secured Dan Taylor as shepherd, thereby bringing to America one of the master Shropshire fitters of the last quarter century. Two years later, he secured Fred Fox and in 1898, Tom Bradburne, who remained with him for over twelve years. Tom possessed the genius for developing through his ovine artisanship what Dr. Davison sought in his capacity as breeder.

The Altamont or Davison type became a distinct stamp in the showyard, arousing violent arguments on more than one occasion. In season and out, Dr. Davison fought and fought for the short-legged, well-sprung type with thickness of back and plumpness of quarters. In his campaign he sent rams to nearly all of the agricultural colleges and experiment stations, to spread the knowledge of, and demand for his sheep. If the college was unable to pay for them, as frequently

happened, he gave them to the institution, thereby starting an acquaintance with his kind of sheep that ultimately won the day in American Shropshire breeding circles.

Altamont rams have had a profound effect in unifying Shropshire standards and in coördinating types throughout all sections of the country. Rams tracing to Borough Magistrate, British Yeoman, or other stock sires in his flock, had a pedigree value in addition to their individual merit, no matter to whom the sale might be. Dr. Davison believed thoroughly that the longer a flock was bred under the same conditions, that is, on the same farm, under the same management, fed by the same feeder, and bred according to the same system, the greater would be the resulting prepotence and uniformity. The lambs of his last show flocks traced back five generations on the sire's side and four on the dam's side to animals of his own breeding, and proved exceptionally strong breeders in other flocks. Altamont sheep were dispersed in 1911, and Dr. Davison thereafter became interested in other fields of agricultural activity.

In 1892, he was elected a director of the Dutchess County (New York) Fair and thus opened a broad career in connection with agricultural exhibitions. During the years that the New York State Fair was held under the auspices of the State Agricultural Society, he was both a member of the Board and secretary and general manager of the show. The last position was held in 1893, while the directorship lasted from 1893 to 1903. When the National Association of Exhibitors of Livestock was organized in 1894, he was elected its secretary, and the following year he was made president of the National Livestock Show at Madison Square Garden, New York. From 1898 to 1914, he was an executive of the American National Livestock Association. In 1899, he became a member of the new association that guaranteed the International Livestock Exposition. He was made a member of the latter's Executive Committee and was its president for the shows of 1917 and 1918. In 1915, he was made a director of the National Horse Show Association of America and, the following year, a director of the Association of American Horse Shows, Inc.

Dr. Davison's permanence of agricultural endeavor is indicated by the variety of organizations for the promotion of live stock and allied interests with which he was identified. He held life memberships in the New York State Agricultural Society, the American Hackney Horse Society and the American Guernsey Cattle Club. For twelve years he was a member of the Executive Committee of the American Shropshire Registry Association, and for three consecutive years was its president. When the American Dairy Shorthorn Association was organized in 1912, Dr. Davison was elected president and served in this capacity for two years. He was a member of the Executive Committee of the National Wool Growers' Association for eight years, and under Governor Levi P. Morton, of New York, was a member of the Board of Control of the New York State Experiment Station at Geneva.

This variety of positions furnished him an opportunity to form an acquaintanceship of national extent, which caused him to drift rather naturally into the journalistic field. He acted as president of the Advanced Agricultural Publishing Company which publishes *The Field Illustrated*; president of the American International Publishers, Inc., which publishes *El Campo International*; and until February 1, 1918, president of the Agricultural Press, Inc., which published *The Agricultural Digest*. In 1916, he was made chairman of the Executive Committee of the National Agricultural Society, which backed the last named publication.

Dr. Davison was born with instincts that made him a lover of the soil and a connoisseur of its products. He was graduated from Yale

University in 1888 and received his bachelor's degree in agriculture from Cornell University one year later. He thereupon entered the American Veterinary College, New York City, where he received his D.V.S. in 1890. His technical training, however, was designed to fit him for a live stock breeder rather than veterinary practice, as he had showed in his boyhood, even as early as the shows of 1879, a tendency toward breeding mastery. His subjects at this time were guinea pigs, and he succeeded in developing squareness of quarters, and carriage of head and crest that made his pigs invincible at the pet stock shows.

The extent and degree of service which Dr. Davison rendered to American agriculture is difficult to estimate. An ardent sportsman, he lent his influence at all times to the upholding and preservation of the sports of rural England, coaching, coursing and the chase. As superintendent of the sheep department of the International, he built up a strong organization that possessed a character fully equivalent to the best of the mutton shows abroad. His example in doing permanent American breeding has served to guide a number of the best sheep breeders since, and so constructive have been their efforts that it has been possible for them to maintain the standards of their respective breeds even when sources of new blood have been denied them through disease quarantines or other handicap.

EDWIN CALLDEMEIER

Dr. Edwin Calldemeier, of Louisville, Ky., was found dead in bed with a bullet wound in his temple, October 12, 1936. He was in his 54th year.

A graduate of the Chicago Veterinary College, class of 1911, Dr. Calldemeier had been in general practice in Louisville for 25 years and had always taken a prominent part in veterinary activities in the Blue Grass State.

Dr. Calldemeier joined the A. V. M. A. in 1916. He was a member of the Committee on Local Arrangements for the Lexington meeting in 1926, and served as Resident Secretary for Kentucky for three years, 1926-28 and 1935-36. He represented his state in the A. V. M. A. House of Representatives at Oklahoma City in 1935. He was a member of Alpha Psi Fraternity and the Twelfth International Veterinary Congress.

PERRY H. BLICKENSTAFF

Dr. Perry H. Blickenstaff died at Pomona, Calif., October 10, 1936. Death was due to carcinoma of the tongue and throat.

Born at Pyrmont, Ind., February 10, 1893, Dr. Blickenstaff attended public schools in Indiana and moved to California in 1907. After attending high school, he entered the State College of Washington for the study of veterinary medicine. He was

graduated in 1923 with the degrees of B. S. and D. V. M., and then engaged in general practice at Chino, Calif., for nine years.

In the fall of 1932, Dr. Blickenstaff went to Ohio State University for a year of postgraduate work. The following year, he received his M. S. degree and returned to California. For a short time, he practiced at Ventura and then accepted a teaching position at the College of Veterinary Medicine, State College of Washington, where he remained until his final illness.

Dr. Blickenstaff joined the A. V. M. A. in 1927. He was a member of the California State Veterinary Medical Association, the Southern California Veterinary Medical Association, the Twelfth International Veterinary Congress, and Chino Lodge No. 373, I. O. O. F. He is survived by his widow (née Hazel Lewis), two sons and his mother.

W. L. C.

MAJOR ARTHUR DUNLAP MARTIN

Major Arthur D. Martin, V. C., U. S. A., died October 23, 1936, at Walter Reed General Hospital, Washington, D. C., after a long illness.

Born in Romulus, N. Y., November 12, 1891, Major Martin attended the Ovid, N. Y., High School and received his veterinary degree from the Indiana Veterinary College in 1915. He was appointed in the regular army, September 7, 1917, with the rank of second lieutenant and was promoted through the grades, attaining his majority September 18, 1931. He served with various stations in the United States and with the A. E. F. in France. He was a graduate of the Army Veterinary School and the Medical Field Service School, and was on duty in the Panama Canal Department at the time of his illness.

Major Martin joined the A. V. M. A. in 1921. He is survived by his widow (née Olga C. Hok) and one son. Services were held in the chapel of the Walter Reed General Hospital, Tuesday morning, October 27, and interment was in Arlington National Cemetery.

WILLIAM R. CLARK

Dr. William R. Clark, of Wauseon, Ohio, died at his home, November 3, 1936, following an illness of about ten days. Death was attributed to cancer of the liver.

Born in Clinton Township, Fulton County, Ohio, November 30, 1862, Dr. Clark received his veterinary training at the Ontario

Veterinary College. Following his graduation in 1898, he was granted certificate number 90 by the Ohio State Board of Veterinary Examiners and located first at Pettisville, Ohio. He was one of the earliest licensed practitioners to locate in northwestern Ohio. In 1899, he removed to Wauseon where he continued in active practice until his fatal illness. Dr. Clark joined the A. V. M. A. in 1928. He was a member of the Northwestern Ohio Veterinary Medical Association.

He is survived by his widow (née Leah Heine), one daughter, one son, and one brother.

H. E. A.

JOHN EDWIN THOMAS

Dr. John E. Thomas, of New Lexington, Ohio, died while visiting an acquaintance in Maxville, Ohio, November 4, 1936. He was 57 years of age and had been a sufferer from a heart ailment for the past two years. Dr. Thomas was a graduate of the Ontario Veterinary College, class of 1907, and had practiced in Perry County for the last 15 years. For several years he was veterinarian to Saint Joseph's Priory, at Somerset, Ohio. He is survived by four sisters and three brothers.

EDWARD JOHNSON EILER

Dr. Edward J. Eiler, of Mount Sterling, Ill., died in a local hospital, November 16, 1936, following an extended illness.

Born at Concord, Ill., September 22, 1871, Dr. Eiler received a grade school education before entering the McKillip Veterinary College. Following his graduation in 1910, he entered general practice and continued in that field until his fatal illness.

Dr. Eiler joined the A. V. M. A. in 1930. He was a member of the Illinois State Veterinary Medical Association. He is survived by his widow (née Ethel Walker), one daughter, one brother and two sisters.

Our sympathy goes out to Dr. Frank R. Butz, of Cincinnati, Ohio, in the death of his wife, November 14, 1936, after an illness of three months, and to Dr. F. F. Dunham, of Vinita, Okla., in the death of his mother, in her 90th year, on November 18, 1936.

PERSONALS

MARRIAGE

DR. C. D. CHASE (K. S. C. '36), of Hart, Mich., to Miss Margaret Wheeler, of Portland, Mich., at Portland, November 7, 1936.

BIRTH

To DR. and MRS. J. F. WITTER, of Orono, Me., a son, Richard Lawrence, September 10, 1936.

PERSONALS

DR. C. L. HAUPERT (O. S. U. '35) is now located at Port Washington, Ohio.

DR. M. A. TAYLOR (Ind. '12), has changed locations from Mattoon to Oakland, Ill.

DR. C. D. CHASE (K. S. C. '36) has removed from Portland, Mich., to Hart, same state.

DR. ELLIOTT PETERSON (Ind. '21) has removed from Vinita, Okla., to San Antonio, Texas.

DR. W. W. LICHTY (Chi. '04), of Woodstock, Ill., has been appointed City Milk Inspector by the City Council.

DR. P. R. BAIRD (Ont. '14), of Waterville, Me., recently completed the remodeling of his small-animal hospital.

DR. OTTO STADER (U. P. '18), who has been in practice at Geneva, Ill., for several years, has located at Ardmore, Pa.

DR. S. C. SHANNON (McK. '17), formerly located at Toluca, and before that at Delavan, has moved to Mackinaw, Ill.

DR. W. M. HENRY (O. S. U. '11), of Jamestown, Ohio, is reported to have purchased two farms recently, of about 360 acres.

DR. PHILLIP A. SOLOMI (O. S. U. '35) operates the Mar Sal Veterinary Hospital, at 12516 Saint Clair Ave., Cleveland, Ohio.

DR. L. E. LONG (Chi. '20), is now employed by the U. S. Bureau of Animal Industry and is engaged in tuberculosis eradication work in Maine.

DR. JOHN E. KELLBERG (U. P. '35), who has been in practice in Chicago for about a year, has located in Princeton, Ill., for general practice.

DR. G. W. LEAHY (McK. '15), of Princeton, Ill., has been appointed Macon County Veterinarian by the Board of Supervisors, effective December 1.

DR. ADOLPH EICHHORN (N. Y.-Amer. '00), of Pearl River, N. Y., has been elected to honorary membership in the Norwegian Veterinary Association.

DR. A. J. NEAL (K. S. C. '17), of Bangor, Me., recently completed the construction of a small-animal hospital, the first of its kind in the city of Bangor.

DR. JAMES E. GRIFFIN (McK. '18), has resigned from the service of the U. S. Bureau of Animal Industry and has entered general practice at Lime Springs, Iowa.